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THE TRANSACTIONS

OF THE

EDINBURGH OBSTETRICAL SOCIETY

THE TRANSACTIONS

OF THE

EDINBURGH OBSTETRICAL SOCIETY.

VOL. XXXIX.

SESSION 1913-1914.

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1914.

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PREFACE.

THIS, the thirty-ninth volume of the Transactions, is a record of the Society's proceedings during the Session 1913-14.

The views expressed are to be taken as the opinions of the Fellows concerned, and do not necessarily reflect the opinion of the Society as a whole.

The Treasurer will be grateful for notices of changes of address, deaths, or any other information necessary to keep the lists of the Society up to date.

THE EDITOR.

October 1914.

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1898 Coe, Prof. Henry C., M.D., 27 East Sixty-fourth St., New York.
1910 Döderlein, Professor Albert Sigmund Gustav, Munich.
1898 Doyen, E., M.D., LL.D., Rue Piccini, 6, Paris.
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1882 Emmet, Dr, 93 Madison Avenue, New York.
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	T. Edgar Underhill, M.D., F.R.C.S. Ed., *Birmingham*,	1879

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	Thomas Dobson Poole, M.D., C.M., *Linthwaite*,	1890
	Alexander William Gordon Price, M.B., C.M.,	1890
	Hugh Shapter Robinson, M.R.C.S. Eng., L.R.C.P. Ed., *London*,	1890
115	Frederick Albert L. Lockhart, M.B., C.M., *Montreal, Canada*,	1890
	Edmund Frederick Tanney Price, M.B., C.M.,	1890
	Owen Foulkes Evans, M.D., C.M., *Liverpool*,	1890
	James Duncan Farquharson, M.B., C.M., *Newcastle-on-Tyne*,	1890
	Harvey Littlejohn, M.B., F.R.C.S. Ed.,	1890
120	Robert Wise, M.D., C.M., *London*,	1890
	William Russell, M.D., F.R.C.P. Ed.,	1890
	*Michael Dewar, M.D., C.M.,	1891
	Gains T. Smith, M.D. *Moncton, New Brunswick*,	1891
	Robert Thin, M.B., F.R.C.P. Ed.,	1891
125	Alexander Henry Vassie, M.B., C.M., *London*,	1891
	James Harvey, M.D., C.M.,	1891
	George Balfour Marshall, M.D., C.M., *Glasgow*,	1891
	William Booth, F.R.C.S. Ed.,	1891
	Richard T. Yoe, M.D., *Louisville, Kentucky, U.S.A.*,	1891
130	Alexander Bruce Giles, M.D., C.M.,	1891
	Hamilton Graham Langwill, M.D., F.R.C.P. Ed., *Leith*,	1891
	Herbert Ernest Lee, M.B., C.M., *Australia*,	1892
	Charles Martin, M.B., C.M., *Kobe, Japan*,	1892
	William Murray Cairns, M.B., C.M., *Liverpool*,	1892

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135	James Thomas Moore Giffen, F.R.C.S. Ed., *Chester*,	1892
	Frank Dendle, M.B., D.P.H., *Isleworth*,	1892
	Simson Carstairs Fowler, M.B., C.M., *Juniper Green*,	1892
	*Prof. John Alexander Campbell Kynoch, M.B., F.R.C.P. Ed., *Dundee*,	1892
	Robert Stirling, M.D., C.M., *Perth*,	1892
140	William Henry Vickery, F.R.C.S. Eng., L.R.C.P. Lond., *Weston-super-Mare*,	1892
	William Ramsay Smith, M.B., C.M., *Australia*,	1892
	John Tod, M.B., C.M., *Leith*,	1892
	George Henry Walter Smith, M.D., C.M., *Sydney, Australia*,	1892
	Charles Cromhall Easterbrook, M.D., C.M., F.R.C.P. Ed., *Dumfries*,	1892
145	Walter Petrie Simpson, M.B., C.M., *Bathgate*,	1892
	*James Ernest Moorhouse, M.D., C.M., *Stirling*,	1892
	*Allen Thomson Sloan, M.D., C.M.,	1893
	Robert Balfour Graham, F.R.C.S. Ed., *Fife*,	1893
	Albert Frederic Rosa, M.D., C.M.,	1893
150	George Benjamin Mitchell, M.B., C.M., *Whitby*,	1893
	John MacRae, M.D., C.M., *Murrayfield*,	1893
	Philip Grierson Borrowman, M.D., C.M.,	1893
	William Herbert Gregory, M.D., C.M., *Beverley, Yorks*,	1893
	James Gibson Cattanach, M.B., F.R.C.P. Ed.,	1893
155	*Alexander Maitland Easterbrook, M.B., C.M., *Gorebridge*,	1893
	Charles Alexander Butchart, M.B., C.M.,	1894
	Frederick Maurice Graham, F.R.C.S. Ed., L.R.C.P. Ed.,	1894
	Robert Hoggan, M.B., C.M., *Liberton*,	1894
	James Livingstone Thompson, M.B., C.M., *Australia*,	1894
160	John Stevens, M.D., F.R.C.P. Ed.,	1894
	Sylvanus Glanville Morris, M.D., C.M., *Mardy*,	1894
	David Robertson Dobie, M.D., C.M., *Crieff*,	1894
	Gopal Govind Vatve, M.D., *Bombay*,	1894
	Robert William Beesley, M.D., C.M., *Bolton*,	1894
165	William A. Stephen, M.D., C.M., *Loftus-in-Cleveland*,	1894
	William Edward Fothergill, M.D., C.M., *Manchester*,	1894
	George Sandison Brock, M.D., C.M., *Rome*,	1894
	*John Martin Munro Kerr, M.B., C.M., F.R.F.P.S.G., *Glasgow*,	1894
	John Montgomery, M.B., C.M., *Birmingham*,	1895
170	*Robert Cochrane Buist, M.D., C.M., *Dundee*,	1895
	Angus Vallance MacGregor, M.D., C.M., *West Hartlepool*,	1895
	John Struthers, M.B., C.M., *Transkei, South Africa*,	1895
	B. W. Broad, M.B., C.M., *Cardiff*,	1895
	Edwin Hindmarsh, M.B., C.M., F.R.C.S. Ed., *Bengal*,	1895
175	Patrick Mackin, M.D., F.R.C.S. Ed., *New Zealand*,	1895
	G. Edgar Helme, M.B., C.M., *Manchester*,	1895
	Percy Theodore Hughes, M.B., C.M., *Broomsgrove*,	1895
	John Hosack Fraser, M.B., F.R.C.P. Ed., *Bridge of Allan*,	1895
	Stewart Grant Ogilvy, M.B., C.M., *Fauldhouse*,	1895
180	Thomas Howard Morgan, M.D., F.R.C.S. Ed., *Queensland, Australia*,	1895
	William Macrae Taylor, M.B., F.R.C.S. Ed.,	1895
	*David James Graham, M.D., F.R.C.P. Ed.,	1895
	Walter William Chipman, M.D., F.R.C.S. Ed., *Montreal*,	1895
	John Cumming, M.D., F.R.C.S. Ed., F.R.C.P. Ed.,	1896
185	Sol Jervois Aarons, M.D., C.M., *London*,	1896
	Robert Beveridge, M.B., C.M., *Leith*,	1896
	John Anderson, M.B., C.M., *Pitlochry*,	1896
	*Robert Gordon M'Kerron, M.B., C.M., *Aberdeen*,	1896
	Frederick John M'Cann, M.D., F.R.C.S. Eng., *London*,	1896

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190	David Robert Taylor, L.R.C.P. & S.Ed., *Ayton*,	1896
	George William Simla Paterson, M.B., C.M.,	1896
	Robert Henry Watson, M.D., C.M., *Cornwall*,	1896
	Thomas Marshall Callender, M.D., C.M., *Sidcup*,	1896
	Lewis Grant, M.D., C.M., *Neston*,	1896
195	Robert Robertson, M.B., C.M.,	1897
	James Wilkie, L.R.C.P. & S. Ed., *Portobello*,	1897
	Andrew Graham, M.D., *Currie*,	1897
	Roderick Murdoch Matheson, M.D., F.R.C.S. Ed.,	1897
	*Robert Jardine, M.D., F.R.F.P.S. Glasg., M.R.C.S. Eng., *Glasgow*,	1897
200	Daniel Charles Edington, M.D., C.M., *Penrith*,	1897
	Harold Sherman Ballantyne, M.B., F.R.C.P.Ed., *Dalkeith*,	1897
	Ernest Edward Porritt, M.D., F.R.C.S. Ed., *New Zealand*,	1897
	William John Garbutt, M.B., C.M., *Birmingham*,	1897
	Henry John Forbes Simson, M.B., F.R.C.S. Ed., *London*,	1897
205	Angus Macdonald, M.B., F.R.C.S. Ed.,	1897
	George Robert Livingston, M.D., C.M., *Dumfries*,	1898
	William Morrison Milne, M.B., C.M.,	1898
	William Joseph Murphy Barry, M.D., M.R.C.P.Ed., *Penarth*,	1898
	Alexander Cruikshank Ainslie, M.D., C.M.,	1898
210	Henry Aylmer Dumat, M.D., F.R.C.P.Ed., *Durban, South Africa*,	1898
	*George Freeland Barbour Simpson, M.D., F.R.C.S. Ed., F.R.C.P. Ed.,	1898
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	James Duncan Slight, M.D., Ch.B., *Leicester*,	1898
215	Francis John Harvey Bateman, M.D., C.M., *London*,	1898
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	Edward William Scott Carmichael, M.D., F.R.C.S. Ed.,	1899
	James Wilson M'Brearty, F.R.C.S. Ed., L.R.C.P. Ed., *New Zealand*,	1899
220	George Crewdson Thomas, M.D., C.M., *London*,	1899
	William John Barclay, M.D., F.R.C.S. Ed., *New Zealand*,	1899
	John Thomas Woodside, L.R.C.P. & S. Ed., *Glamorganshire*,	1900
	Owen, St John Moses, M.D., C.M., B.Sc., *India*,	1900
	*Harry Oliphant Nicholson, M.D., F.R.C.P. Ed.,	1900
225	Thomas Scott Brodie, M.B., C.M., *Wishaw*,	1900
	William Hope Fowler, M.B., Ch.B.,	1900
	Ogden Watson Ogden, M.D., M.R.C.S., *Newcastle-on-Tyne*,	1900
	John Craig, M.B., Ch.B.,	1900
	William Hartley Bunting, M.D., F.R.C.S. Ed., *Birmingham*,	1900
230	Theodore Charles Mackenzie, M.B., Ch.B., *Inverness*,	1900
	John Boyd Jamieson, M.D., F.R.C.S. Ed.,	1900
	Malcolm M'Larty, M.B., C.M.,	1900
	Frederick Gardiner, M.D., C.M.,	1900
	George Mackie, M.B., Ch.B., *Malvern*,	1900
235	*Kenmure Duncan Melville, M.D., Ch.B.,	1900
	John Thomas Dickie, L.R.C.P. & S. Ed.,	1900
	*William Ernest Frost, M.B., Ch.B.,	1900
	*Frederick David Simpson, M.D., F.R.C.S. Ed.,	1900
	William Darling, M.B., F.R.C.S. Ed.,	1900
240	Malcolm Campbell, M.B., F.R.C.S. Ed., *Brechin*,	1900
	James Ramsay Munro, M.D., Ch.B., *Spalding*,	1901

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	George James Rogerson Carruthers, M.B., Ch.B.,	1901
	Hilda Maud M'Farlane Sturrock, L.R.C.P. & S. Ed., *Perth*,	1901
	George Dickson, M.D., C.M.,	1901
245	Elsie Maud Inglis, M.B., C.M.,	1901
	George Robertson, L.R.C.P. & S. Ed., *Dunfermline*,	1901
	John Wishart Kerr, M.B., Ch.B., *Glasgow*,	1901
	William Harold Graham Aspland, M.D., M.R.C.S. Eng., *China*,	1901
	William Taylor McArthur, M.D., F.R.C.S. Ed., *California*,	1901
250	*Robert Patton Ranken Lyle, M.D., Ch.B., *Newcastle-on-Tyne*,	1901
	Kennedy C. M'Ilwraith, M.B., M.C.P. & S. Ont., *Toronto*,	1901
	Robert Alexander John Harper, M.D., Ch.B., *Dalkeith*,	1901
	William Hogg Prentice, M.D., Ch.B., *Pendleton*,	1901
	*Alexander Mowatt Malcolmson, M.D., Ch.B., *Corstorphine*,	1901
255	Caleb Williams Saleeby, M.D., Ch.B., *London*,	1902
	John Ligertwood Green, M.D., Ch.B.,	1902
	William Sloss, M.B., Ch.B., *Australia*,	1902
	Charles James Hill Aitken, M.D., C.M., *Cape Colony*,	1902
	E. R. Secord, M.D., *Ontario*,	1902
260	F. E. Thompson, M.D., *Montreal*,	1902
	John M'Gibbon, M.B., F.R.C.P.Ed.,	1902
	Thomas James Thomson, M.D., C.M.,	1902
	Charles Mowbray Pearson, M.B., F.R.C.P.Ed.,	1902
	Ewen John Maclean, M.D., M.R.C.P. Lond., *Cardiff*,	1902
265	James William Somerville, M.D., C.M., *Galashiels*,	1902
	Alexander Miller, L.R.C.P. Ed., L.F.P.S. Glasg., *Glasgow*,	1902
	Duncan Macnab Callender, M.B., Ch.B., *Lancaster*,	1902
	Robert Cranston Low, M.B., F.R.C.P.Ed.,	1902
	Benjamin Philp Watson, M.D., F.R.C.S.Ed., *Toronto*,	1902
270	John Macdonald, M.B., C.M., *Cupar-Fife*,	1902
	Charles William Somerville, M.B., Ch.B., *China*,	1902
	Frank Mayes Willcox, M.B., C.M.,	1902
	John Tennant, M.B., C.M., *Scunthorpe*,	1902
	Alexander Simpson Wells, M.B., F.R.C.S. Ed., *Cape Town*,	1903
275	Andrew Binny Flett, M.B., Ch.B.,	1903
	William Henry Eden Brand, F.R.C.S. Ed., L.R.C.P. Ed., *Banchory*,	1903
	Alfred Lambré White, L.R.C.P. & S. Ed., *Manchester*,	1903
	Robert Bathgate Johnston, L.R.C.P. & S. Ed., *Penrith*,	1903
	Robert Wilson Gibson, M.D., F.R.C.S. Ed., *Orton*,	1903
280	Philip Henry Mules, M.B., Ch.B., *New Zealand*,	1903
	Andrea Francis Honyman Rabagliati, M.D., Ch.B., *Bradford*,	1903
	Donald Gregor MacArthur, M.D., C.M., *London*,	1903
	Henry Martyn Stumbles, M.D., Ch.B., *Sussex*,	1903
	Gilbert John Farie, M.B., Ch.B., *London*,	1903
285	Katherine Jane Stark Clark, M.D., Ch.B., D.P.H.,	1903
	David Halliday Croom, M.D., Ch.B.,	1903
	Eleanor Russell Elder, M.B., Ch.B., *Leith*,	1903
	*Robert William Johnstone, M.D., F.R.C.S.Ed.,	1903
	*James William Keay, M.D., Ch.B.,	1903
290	Ivan Cochrane Keir, M.D., Ch.B., *Melksham*,	1903
	Alexander Grant Macdonald, M.B., C.M., *Isle of Skye*,	1903
	Charles John Shaw, M.D., Ch.B., *Lochgilphead*,	1903
	*Frederick Porter, M.B., C.M.,	1904

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	Russell Gerald William Adams, M.D., Ch.B., *New Zealand*,	1904
295	Duncan Campbell Lloyd Fitzwilliams, M.D., F.R.C.S.Ed., *London*,	1904
	Clarence Brian Dobell, M.B., M.R.C.S. Eng., M.R.C.P.L., *Cheltenham*,	1904
	Hugh Stevenson Davidson, M.B., Ch.B.,	1904
	Andrew Milroy Fleming, C.M.G., M.B., F R.C.S. Ed., *Rhodesia*,	1904
	Thomas William Edmonston Ross, M.B., Ch.B., *Cardiff*,	1904
300	John Benjamin Hellier, M.D., M.R.C.S. Eng., *Leeds*,	1904
	William Brown, M.B., Ch.B., *Aberdeen*,	1904
	Henry Howard Robarts, M.D., Ch.B., *Haddington*,	1904
	Robert Balfour Barnetson, M.B., Ch.B., *Portobello*,	1904
	James Lochhead, M.D., F.R.C.S.Ed.,	1904
305	Arthur Charles Strain, M.D., Ch.B., *West Hartlepool*,	1904
	Lewis Beesly, L.R.C.P.Ed., F.R.C.S. Ed.,	1904
	James Crawford Gibb Macnab, M.B., F.R.C.S. Ed.,	1904
	Robert William Lessel Wallace, M.B., Ch.B., *Bagdad*,	1905
	Alastair MacGregor, M.D., C.M., *London*,	1905
310	Edmond Frost, M.D., C.M., *Eastbourne*,	1905
	Edith Cochrane-Brown Pitts, M.B., Ch.B., *New Zealand*,	1905
	James Brownlee, M.D., Ch.B., *Middlesbrough*,	1905
	Peter M'Ewan, M.B., Ch.B., *Bradford*,	1905
	George Douglas Mathewson, M.B., Ch.B.,	1905
315	Andrew Fleming, M.D., Ch.B., *Corstorphine*,	1905
	Robert William Craig, M.D., Ch.B., *Ford*,	1905
	Henry John Dunbar, M.D., Ch.B., *Cardiff*,	1905
	Richard Alfred Blake, M.D., Ch.B., *Transvaal*,	1905
	John Herbert Gibbs, F.R.C.S.Ed.,	1905
320	Alice Marion Hutchison, M.D., Ch.B.,	1905
	W. T. Chenhall, M.D., *Sydney*,	1906
	Alexander Angus Martin, F.R.C.S.Ed., *North Shields*,	1906
	James Andrew Gunn, M.D., Ch.B.,	1906
	James Lawson Russell, M.B., Ch.B., *Todmorden*,	1906
325	Archibald M'Kendrick, F.R.C.S. Ed., L.R.C.P. Ed.,	1906
	Alexander Scott, M.B., C.M., *Broxburn*,	1906
	Archibald Simpson, M.B., Ch.B., *Tranent*,	1906
	Hirjee Nowroje Anklesaria, L.R.C.P. & S.Ed., *Bombay*,	1906
	Archibald Cotterell M'Master, M.B., F.R.C.S.Ed., *New Zealand*,	1906
330	Herbert Pank Thompson, M.D., F.R.C.S. Ed., *Harrogate*,	1906
	Samuel Davidson, M.D., C.M., *Kelso*,	1906
	William Fowler Godfrey, M.B., C.M., *Manitoba*,	1906
	Edward Burnet, M.B., Ch.B. *Leamington*,	1906
	Arthur Samuel Walker, M.B., Ch.B., *Leith*,	1906
335	Archibald Dunlop Stewart, M.B., L.R.C.S. Ed.,	1906
	Edward Alexander Elder, M.B., F.R.C.S.Ed.,	1906
	Ethelbert William Dyer, M.B., F.R.C.S.Ed., *Durban*,	1906
	William Torrance Smith, M.B., Ch.B., *Liverpool*,	1906
	Arnold Davies, M.B., Ch.B., *Menai Bridge*,	1906
340	John Bruce M'Moreland, M.B., Ch.B., *Moniaive, Dumfriesshire*,	1906
	William Omand Sclater, M.B., Ch.B., *Perak*,	1906
	Archibald George Kirkwood Ledger, M.D., Ch.B., *Coppull*,	1906
	Frederick James Greig, L.R.C.P. & S.I., Lt.-Col., R.A.M.C., *Stirling*,	1906
	Duncan Lorimer, M.B., F.R.C.S.Ed.,	1906

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345	Charles Robert Paterson Mitchell, M.B., Ch.B., *Malvern*,	1906
	John Halley Meikle, M.D., D.P.H.,	1906
	David Lloyd Roberts, M.D., F.R.C.P., *Manchester*,	1906
	Elsie Mary Macmillan Barnetson, M.B., Ch.B., *Joppa*,	1907
	John Andrew Macleod, M.B., Ch.B., *Inverness*,	1907
350	Hugh Smith Reid, M.D., F.R.C.S.Ed.,	1907
	Angus Buchanan, M.B., Ch.B.,	1907
	William Bow Tannahill, M.B., Ch.B., *Greenock*,	1907
	Aubrey Middleton Hewat, M.B., Ch.B., *Preston*,	1907
	Isabella Beattie Venters, M.B., Ch.B.,	1907
355	Gerald Hall Lloyd Fitzwilliams, M.B., F.R.C.S.Ed., *Newcastle-Emlyn*,	1907
	Ronald Græme Scott Orbell, M.D., Ch.B., *New Zealand*,	1907
	John Alexander Gray, M.B., Ch.B.,	1907
	Cecil Hobart Houghton, M.B., Ch.B., *Cape Colony*,	1907
	Robert Patrick M'Morland, M.B., Ch.B., *Dumfries*	1907
360	James Young, M.D., F.R.C.S.Ed.,	1907
	John Nigel Stark, M.D., F.R.F.P.S.G., *Glasgow*,	1908
	Norman Scott Carmichael, M.B., Ch.B.,	1908
	Harley Patterson Milligan, M.D., F.R.C.S.Ed., *Hessle*,	1908
	Thomas Yule Finlay, M.B., Ch.B.,	1908
365	David Lawrence Luckhoo, M.D., L.R.C.P.S.Ed., *British Guiana*,	1908
	James Crawford Craig, M.B., Ch.B., *London*,	1908
	John Gibson Craig, M.B., F.R.C.S.Ed., *York*,	1908
	Alexander Gibson, M.B., Ch.B.,	1908
	Hugh Ruthven Lawrence, M.D., Ch.B., *Cape Colony*,	1908
370	Alexander Cross Mallace, M.B., Ch.B., *Culross*,	1908
	John More, L.R.C.P.Lond., M.R.C.S.Eng., *Rothwell*,	1908
	Percy Wilfred Shepherd, M.B., C.M., *Leeds*,	1908
	David Elliot Dickson, M.D., F.R.C.S.Ed., *Lochgelly*	1908
	John M'Culloch, M.D., C.M., *Ontario*,	1909
375	Robert Brodie Anderson, M.D., F.R.C.S.Ed., *Manitoba*,	1909
	George Scott Carmichael, M.D., Ch.B., M.R.C.P.E.,	1909
	William James Crow, M.B., Ch.B.,	1909
	Alfred Edward Turnbull, M.B., Ch.B., *Berks*,	1909
	Alexander Aitken Ross, M.B., Ch.B.,	1910
380	Stephen Forrest, M.D., Ch.B.,	1910
	Herbert Lindesay Watson Wemyss, M.D., Ch.B.,	1910
	John M'Laren, M.B., C.M.,	1910
	William Fraser Macdonald, M.B., Ch.B.,	1910
	Morgan Howard Howard-Jones, L.R.C.P. & S. Ed.,	1910
385	R. C. Alexander, M.B., Ch.B.,	1910
	Keith B. MacGlashan, M.B., Ch.B., *Leeds*,	1910
	Matthew R. Drennan, M.A., M.B., Ch.B.,	1910
	F. E. Jardine, M.B., Ch.B., *Penicuik*,	1910
	R. S. Mowat, M.D.	1910
390	W. W. Carlow, M.B., Ch.B.,	1910
	J. M. Dewar, M.B., Ch.B.,	1910
	A. J. Gordon Hunter, M.B., Ch.B.,	1910
	W. Menzies Menzies, M.B., Ch.B.,	1910
	David Shannon, M.B., Ch.B., *Glasgow*,	1910
395	Lewis H. F. Thatcher, M.D., M.R.C.P.E.,	1910
	A. L. S. Tuke, M.B., C.M., *Dunfermline*,	1911
	James Bruce Ronaldson, M.R.C.S., L.R.C.P., *Bucks*,	1911
	James Langwill, M.B., Ch.B., *Joppa*,	1911
	P. J. Olivier, M.B., Ch.B.,	1911
400	T. C. Ritchie, M.B., Ch.B., *Glasgow*,	1911
	D. R. B. Sivright, B.A., M.B., Ch.B., *Canterbury*,	1911

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	A. E. Chisholm, M.B., Ch.B., F.R.C.S.Ed.,	1911
	H. Gordon Chouler, L.R.C.P., &c., L.D.S.,	1911
	Wm. Maitland Hume, M.D., F.R.C.S.E., *Perth*,	1911
405	Robert Abernethy, M.D., F.R.C.P.E.,	1912
	William Anderson, M.B Ch.B., *Armadale, Linlithgowshire*,	1912
	Alfred Jordan Beattie, M.B., Ch.B., *Callander*,	1912
	Arthur James Beveridge, M.B., C.M., *Dollar*,	1912
	L. F. Bianchi, F.R.C.S.E., *Joppa*,	1912
410	J. M. Bowie, M.D., F.R.C.S.E.,	1912
	D. C. Bremner, M.B., C.M., *Peebles*,	1912
	A. J. Brock, M.D.,	1912
	W. S. Murdoch Brown, M.B., Ch.B.,	1912
	Arthur Burton, M.D. (Cantab.), *Cromer*,	1912
415	James Butler, M.B., C.M., *Leith*,	1912
	E. Percy Calder, M.B., Ch.B., *Coldingham*,	1912
	A. N. S. Carmichael, M.B., C.M.,	1912
	Arthur W. Stark Christie, M.B., F.R.C.S.E., *Doune*,	1912
	Andrew Croll, M.D., *Saskatchewan, Canada*,	1912
420	George A. Dickson, M.B., C.M., *South Queensferry*,	1912
	George Donald, M.D., *Leith*,	1912
	Charles Hotham Evans, M.R.C.S., L.R.C.P., *London, W.*,	1912
	Thomas Finlay, M.B., C.M.,	1912
	L. P. M. Gardner, M.B., Ch.B., *Leith*,	1912
425	A. Fergus Hewat, M.B., Ch.B.,	1912
	David Huskie, M.B., C.M., *Moffat*,	1912
	W. Wilton Johns, M.D., *Nairn*,	1912
	G. Minto Johnston, M.D., *Leith*,	1912
	K. B. Kanga, B.Sc., L.M.R.C.P. Ireland, L.M. Dublin, *Bombay, India*,	1912
430	F. K. Kerr, M.B., Ch.B., *Leith*,	1912
	H. A. Leebody, M.B., C.M., *Corstorphine*,	1912
	W. Leslie, M.D., F.R.C.S.E., *Alloa*,	1912
	A. Morrison M'Intosh, M.B., F.R.C.S.E.,	1912
	Malcolm E. Mackay, M.D., C.M. (M'Gill), *Saskatchewan, Canada*,	1912
435	Robert M'Laren, L.R.C.P.E., etc.,	1912
	L. R. H. P. Marshall, M.D., *Peebles*,	1912
	Angus Matheson, M.D., *North Berwick*,	1912
	George Melville, M.B., C.M., *Penicuik*,	1912
	A. S. Miller, M.D., *Tranent*,	1912
440	Alexander Nasmyth, M.D., *Penicuik*,	1912
	John Orr, M.D., F.R.C.P.E.,	1912
	T. S. A. Orr, M.B., Ch.B., *Crail*,	1912
	William Paterson, M.B., C.M., *Leith*,	1912
	George R. Petersen, M.B., C.M. (M'Gill). *Saskatchewan, Canada*,	1912
445	George B. Ritchie, M.B., C.M.,	1912
	R. Macdonald Robertson, M.D.,	1912
	R. Miller Ronaldson, M.D., F.R.C.P.E.,	1912
	H. Basil Rosair, L.M.R.C P.I., L.R.C.S.I., etc., *London*,	1912
	Behram Pestonjee Sabawala, M.R.C.S., L.R.C.P. Lond., *Bombay, India*,	1912
450	W. J. Burns Selkirk, M.D., *Dunbar*,	1912
	T. R. S. Sibbald, M.D., *Perthshire*,	1912
	John W. L. Spence, L.R.C.P.E.,	1912
	James A. Taylor, M.D., *Dunkeld*,	1912
	Alexander Veitch, M.B., C.M.,	1912
455	A. Murray Wood, M.D., *Leith*,	1912
	D. M'Farlane Barker, M.B., Ch.B.,	1912
	Alexander J. W. Buchanan, M.B., Ch.B., *Cockenzie*,	1912
	Morton Burnet, M.D., *Crieff*,	1912

		Date of Admission.
	William L. Cullen, M.B., C.M., *St Boswells*,	1912
460	Samuel Fergusson, M.B., C.M., *Alloa*,	1912
	James T. Gunn, M.B., F.R.C.S.E., *Auchterarder*,	1912
	Richard Heard, M.B., Ch.B., B.A.O., R.U.I., Major I.M.S., *London, S.W.*,	1912
	John Honeyford, M.B., Ch.B.,	1912
	Thomas Harcourt Lawrie, L.R.C.P.E., etc., *Polmont Station, Stirlingshire*,	1912
465	Alexander M'Ewen, M.B., C.M., *Crieff*,	1912
	George M'Kellar, M.D., *Kelso*,	1912
	A. G. Ritchie, B.Sc., M.B., Ch.B.,	1912
	Stanley F. Silberbauer, M.B., Ch.B., *Cape Town, South Africa*,	1912
	Murray B. Steuart, M.B., C.M., *Kirkcudbright*,	1912
470	Ernest A. Walker, M.B., Ch.B., Captain I.M.S., *London*,	1912
	Norman P. Fairfax, L.R.C.P., etc., *Innerleithen*,	1912
	Hermann Kramer, M.D., F.R.C.S.,	1912
	Laura Stewart-Sandeman, M.D., *Aberdeen*,	1912
	Peter Stewart, L.R.C.P., etc., *Uphall*,	1912
475	Anne Mercer Watson, L.R.C.P., etc., *Aberdeen*,	1912
	Edward Napier Burnett, M.D. (Glasg.), F.R.C.S.Ed., *Newcastle-on-Tyne*,	1912
	Basil Patrick Campbell, B.A., M.B. (Cantab), F.R.C.S.E., *Brechin*,	1912
	George Maxwell Elliott, M.B., Ch.B.,	1912
	A. C. Hendrick, M.A., M.B. (Toronto), F.R.C.S.Ed., *Toronto*,	1912
480	Alexander Hutchison, M.B., Ch.B., *Grantown-on-Spey*,	1912
	Archibald Alexander Morison, M.B., Ch.B.,	1912
	John H. H. Pearson, M.B., Ch.B.,	1912
	F. A. Scannell, M.B., Ch.B. (N.Z.),	1912
	A. C. Thomson, M.B., Ch.B. (N.Z.),	1912
485	Alexander E. Watson, M.B., C.M., *East Wemyss*,	1912
	Robert Honey Watt, M.D., *Langholm*,	1912
	Tom Welsh, M.B., Ch.B.,	1912
	Gilbert Vere Bogle, M.B., Ch.B.Ed., *New Zealand*,	1912
	George Selby Brock, M.B., Ch.B.Ed., L.M., *Bombay*,	1912
490	Maurice Cameron Anderson, L.R.C.P.E., L.R.C.S.E., L.F.P.S.G., *Roslin*,	1913
	Robert Wilson Buchanan, M.A., B.Sc., M.B., Ch.B.,	1913
	C. Lodge Patch, M.B., Ch.B., *Paisley*,	1913
	H. W. Webb, M.B., Ch.B.,	1913
	Robert Crothers, F.R.C.S., *Brighton*,	1913
495	Gordon Fitzgerald, M.D. Ed., *Manchester*,	1913
	T. W. J. Childs, M.B., Ch.B.,	1913
	Thomas Wm. Clarke, M.B., Ch.B., *Sheffield*,	1913
	H. J. Godwin, M.B., F.R.C.S. Ed., *Winchester*,	1913
	Archibald Langwill, M.B., Ch.B.,	1913
500	Frank Grégoire Proudfoot, M.D., *Oxford*,	1913
	A. M. Stewart, M.B., Ch.B., *Leith*,	1913
	J. D. Grahame Stewart, M.B., Ch.B.,	1913
	J. R. Jackson Hartley, M.B., Ch.B.,	1914
	A. G. M. Grant, M.B., Ch.B., *Newtown, North Wales*,	1914
505	D. T. Guthrie, M.B.,	1914
	Patrick A. Bennet Clark, M.B., Ch.B.,	1914
	J. H. Graham Hunter, M.B., Ch.B.,	1914
	John L. Annan, M.B., Ch.B.,	1914
	T. Douglas Inch, M.B., Ch.B., *Gorebridge*,	1914
510	J. W. Pell, L.R.C.S. & P. Ed.,	1914
511	Ernest Muir, M.D.,	1914

ORDINARY FELLOWS.

ARRANGED ALPHABETICALLY.

(*a.*) LIFE MEMBERS.

		Date of Admission.
	Aarons, Dr S. Jervois, 6 Hartley Street, Cavendish Square, London, W.,	1896
	Adam, Dr George Rothwell, 84 Collins Street, Melbourne, Australia,	1879
	Anderson, Dr John, Newholme, Pitlochry,	1896
	Ballantyne, Dr Harold S., Ashton, Eskbank, Dalkeith,	1897
5	Ballantyne, Dr J. W., 19 Rothesay Terrace,	1883
	Barbour, Dr A. H. Freeland, 4 Charlotte Square,	1879
	Barclay, Dr William John, Invercargill, New Zealand,	1899
	Barrington, Dr Fourness, 213 Macquarrie Street, Sydney, Australia,	1884
	Brock, Dr G. Sandison, 6 Corso d'Italia, Rome,	1894
10	Brock, Dr George Selby, c/o Grindlay, Groom & Co., Bombay,	1913
	Burnet, Dr Edward, Dalblair, Lillington Road, Leamington,	1906
	Burton, Dr Arthur, Stenson, Cromer,	1912
	Chenhall, Dr William T., 233 Macquarie St., Sydney, Australia,	1906
	Chipman, Dr W. W., 285 Mountain St., Montreal, Canada,	1895
15	Craig, Dr John, 216 Bruntsfield Place,	1900
	Craig, Dr William, 71 Bruntsfield Place,	1870
	Croom, Dr David Halliday, 17 Alva St.,	1903
	Croom, Prof. Sir John Halliday, 25 Charlotte Square,	1870
	Cullen, Dr William L., Fairholme, St Boswells,	1912
20	Cumming, Dr John, 70 Bruntsfield Place,	1896
	Davidson, Dr Hugh, 6 Atholl Place,	1904
	Dobell, Dr C. B., 1 Royal Well Terrace, Charlton, Cheltenham,	1904
	Dumat, Dr Henry Aylmer, 7 Devonshire Place, Durban, Natal, South Africa,	1898
	Ferguson, Dr J. Haig, 7 Coates Crescent,	1885
25	Fitzwilliams, Dr Gerald Lloyd, Cilgwyn, N.C. Emlyn, R.S.O., Carmarthenshire,	1907
	Fleming, Dr Andrew M., C.M.G., Salisbury, Rhodesia,	1904
	Fowler, Dr Simson, Waverley, Juniper Green,	1892
	Frost, Dr Edmund, Chesterfield, Meads, Eastbourne,	1905
	Gibson, Dr R. Wilson, Town Head House, Orton, Tebay,	1903
30	Grant, Dr Lewis, Neston, Cheshire,	1896
	Hart, Dr D. Berry, 5 Randolph Cliff,	1877
	Haultain, Dr F. W. N., 12 Charlotte Square,	1884
	Houghton, Dr C. Hobart, Fort Hare, Alice, Cape Colony, South Africa,	1907
	Hutchison, Dr Alex., Stonefield, Grantown-on-Spey,	1913
35	Inch, Dr Robert, Gorebridge,	1887
	Johns, Dr W. Wilton, Ramleh, Nairn,	1912
	Kanga, Dr K. B., Kanga Building, Khetmadi Back Road, Bombay, India,	1912
	Kerr, Dr F. K., 168 Newhaven Road, Leith,	1912
	Livingston, Dr George R., 47 Castle Street, Dumfries,	1898
40	Luckhoo, Dr David Lawrence, New Amsterdam, British Guiana,	1908
	M'Arthur, Dr W. Taylor, 2025 Western Avenue, Los Angeles, California,	1901

		Date of Admission.
	M'Brearty, Dr J. Wilson, Greymouth, West Coast, New Zealand,	1899
	M'Culloch, Dr John, Lindsay, Ontario, Canada,	1909
	M'Master, Dr A. C., Erinholm, Hopetoun St., Auckland, New Zealand,	1906
45	Macnab, Dr James C. G., Germiston, Transvaal, South Africa	1904
	Maddox, Dr Ralph H., I.M.S., c/o Messrs Thomas Cook & Son, Ludgate Circus, London, E.C.,	1887
	Martin, Dr Christopher, Cleveland House, George Road, Edgbaston, Birmingham,	1888
	Melville, Dr George, The Eyrie, Penicuik,	1912
	Melville, Dr Kenmure, 2 Nile Grove,	1900
50	Morgan, Dr T. H., Gympie, Queensland, Australia,	1895
	Mules, Dr P. Henry, Woodville, Wellington, New Zealand,	1903
	Pereira, Dr A. A. Jervis, Physician to the Mozambique Company, Beira, East Africa,	1888
	Pitts, Dr Edith Cochrane-Brown, Strathmore, Christ Church, New Zealand,	1905
	Ranking, Dr J. E., Tunbridge Wells,	1881
55	Ritchie, Dr A. G., 22 Charlotte Square,	1912
	Ronaldson, Dr J. Bruce, Penn Road, Beaconsfield, Bucks,	1911
	Ross, Dr James F. W., 481 Sherbourne Street, Toronto, Canada,	1889
	Russell, Dr J. Lawson, West Lodge, Todmorden,	1906
	Sabawala, Dr Behram Pestonjee, c/o Messrs King, King & Co., Hornby Road, Bombay, India,	1912
60	Shepherd, Dr Percy W., Sydenham House, Pontefract Lane, Leeds,	1908
	Simpson, Dr G. F. Barbour, 43 Manor Place,	1898
	Simpson, Dr W. Petrie, Viewbank, Bathgate,	1892
	Simson, Dr H. J. F., 36 Grosvenor Street, London, W.,	1897
	Sivright, Dr D. R. B., Kent and Canterbury Hospital, Canterbury,	1911
65	Sloss, Dr William, Sturt Street, Ballarat, Australia,	1902
	Stewart, Dr J. D. Grahame, Maternity Hospital,	1913
	Struthers, Dr John, Nqamakwe, Transkei, South Africa,	1895
	Sturrock, Dr Hilda M., Northcote, Edinburgh Road, Perth,	1901
	Taylor, Dr James A., Dunkeld,	1912
70	Vatve, Dr Gopal Govind, 42 Churin Road, Girgaon, Bombay, India,	1894
	Wells, Dr A. Simpson, 118 New Church Street, Cape Town, South Africa,	1903

(*b.*) ANNUAL SUBSCRIBERS.

	Abernethy, Dr Robert, 10 St Colme Street,	1912
	Adams, Dr Russell G. W., Maxwell Road, Blenheim, New Zealand,	1904
	Affleck, Sir J. O., 38 Heriot Row,	1869
75	Ainslie, Dr A. C., 49 Minto Street,	1898
	Aitken, Dr C. J. Hill, Idutywa, Transkéi, Cape Colony, South Africa,	1902
	Alexander, Dr R. C., 15 Hartington Gardens,	1910
	Alexander, Dr W. B., 8 Blenheim Place,	1882
	Allison, Dr J., Fuller House, Kettering, Northampton,	1888
80	Anderson, Dr Maurice Cameron, Roslin, Midlothian,	1913
	Anderson, Dr R. Brodie, F.R.C.S.Ed., 666 Main Street, Winnipeg, Manitoba, Canada,	1909
	Anderson, Dr William, Armadale, Linlithgowshire,	1912

		Date of Admission.
	Anklesaria, Dr H. N., Taj Building, Hornby Road, Bombay, India,	1906
	Annan, Dr John L., 27 Braid Road,	1914
85	Armitage, Dr J. A., Netherwood, St Helens Park, Hastings,	1886
	Armour, Dr E. F., 6 Bruntsfield Terrace,	1889
	Aspland, Dr W. H. Graham, Church of England Mission, Peking, China,	1901
	Baildon, Dr Francis J., 42 Hoghton Street, Southport,	1887
	Ballantyne, Dr A., Oxfordholme, Gullane,	1870
90	Barker, Dr D. M'Farlane, 4 Dalziel Place,	1912
	Barnetson, Dr Elsie M., Woodville, Joppa,	1907
	Barnetson, Dr R. Balfour, Woodville, Joppa,	1904
	Barry, Dr W. J. M., 29 Plymouth Road, Penarth, Glamorgan,	1898
	Bateman, Dr F. J. Harvey, Heath End, Blackheath, Lond., S.E.,	1898
95	Beattie, Dr Alfred Jordan, Riverview, Callander,	1912
	Beatty, Dr Samuel, Craigvar, Pitlochry,	1888
	Beesley, Dr R. W., 135 Deane Road, Bolton,	1894
	Beesly, Dr Lewis, 42 Northumberland Street,	1904
	Bell, Dr J. Lumsden, The White Hall, Driffield, Yorkshire,	1884
100	Beveridge, Dr Arthur James, Holmlea, Dollar,	1912
	Beveridge, Dr Robert, 9 James Place, Leith,	1896
	Bianchi, Dr L. F., 40 Brighton Place, Portobello,	1912
	Blaikie, Dr R. H., 5 Mayfield Gardens,	1888
	Blair, Dr J. A., 16 Windsor Terrace, Newcastle-on-Tyne,	1887
105	Blake, Dr R. A., Belfast, Transvaal, South Africa,	1905
	Boddie, Dr G. P., 30 Coates Gardens,	1889
	Bogle, Dr Gilbert Vere, Waipukurau, New Zealand,	1912
	Booth, Dr William, 2 Minto Street,	1891
	Borrowman, Dr Philip G., 74 Great King Street,	1893
110	Bowie, Dr J. M., 53 Grange Road,	1912
	Brand, Dr Eden, Bellfield, Banchory,	1903
	Bremner, Dr D. C., Hollybush, Peebles,	1912
	Brewis, Dr N. T., 6 Drumsheugh Gardens,	1883
	Brewis, Dr R. Adams, The West Gate, Dursley, Gloucestershire,	1888
115	Broad, Dr B. W., The Sanitorium, Cardiff,	1895
	Brock, Dr A. J., 27 Braid Crescent,	1912
	Brodie, Dr T. Scott, 21 Belhaven Terrace, Wishaw,	1900
	Brown, Dr William, 7 Bonaccord Square, Aberdeen,	1904
	Brown, Dr W. S. Murdoch, 13 Torphichen Street,	1912
120	Brownlee, Dr James, Waterloo House, Linthorpe Road, Middlesbrough,	1905
	Buchanan, Dr Alexander J. W., Ruth Villa, Cockenzie,	1912
	Buchanan, Dr Angus, 45 Falcon Avenue,	1907
	Buchanan, Dr Robert Wilson, 5 Granville Terrace,	1913
	Buist, Dr J. W., 1 Clifton Terrace,	1877
125	Buist, Dr R. C., 166 Nethergate, Dundee,	1895
	Bunting, Dr W. Hartley, 20 Hagley Road, Edgbaston, Birmingham,	1900
	Burnet, Dr Morton, Beechknowe, Crieff,	1912
	Burnett, Dr Edward Napier, 4 Windsor Crescent, Newcastle-on-Tyne,	1912
	Butchart, Dr C. A., 52 Leith Walk, Leith,	1894
130	Butler, Dr James, 49 Charlotte Street, Leith,	1912
	Cairns, Dr W. Murray, 67 Catharine Street, Liverpool,	1892
	Calder, Dr E. Percy, Kirkhill, Coldingham,	1912
	Calder, Dr H. L., 60 Leith Walk, Leith,	1882
	Callender, Dr D. M., 12 Rose Bank, Lancaster,	1902
135	Callender, Dr T. M., Inverard, Sidcup,	1896
	Campbell, Dr Basil Patrick, 28 Panmure Street, Brechin,	1912

		Date of Admission.
	Campbell, Dr Malcolm, Westwood, Brechin,	1900
	Carlow, Dr W. W., 22 Melville Street,	1910
	Carmichael, Dr A. N. S., 2 Merchiston Avenue,	1912
140	Carmichael, Dr Edward, 21 Abercromby Place,	1887
	Carmichael, Dr E. W. Scott, 40 Palmerston Place,	1899
	Carmichael, Dr George S., 25 Braid Road,	1909
	Carmichael, Dr Norman Scott, 44 Polwarth Terrace,	1908
	Carruthers, Dr G. J. R., 4 Melville Street,	1901
145	Cattanach, Dr J. G., 3 Alvanley Terrace,	1893
	Childs, Dr T. W. J., Royal Maternity Hospital, Lauriston Place,	1913
	Chisholm, Dr A. E., 7 Claremont Crescent,	1911
	Chouler, Dr H. Gordon, University Hall, 2 Mound,	1911
	Christie, Dr Arthur W. Stark, Balkerach, Doune,	1912
150	Church, Dr H. M., 28 Cluny Drive,	1875
	Clark, Dr Katherine S., 16 Drummond Place,	1903
	Clark, Dr Patrick A. Bennet, 15 Douglas Crescent,	1914
	Clarke, Dr Thomas William, The Infirmary, Sheffield,	1913
	Cox, Dr Joshua J., 72 Bridge Street, Manchester,	1876
155	Craig, Dr James C., 113 Albany Street, Regent's Park, London, N.W.,	1908
	Craig, Dr J. G., 78 Bootham, York,	1908
	Craig, Dr R. W., Pathhead-Ford, Dalkeith,	1905
	Croll, Dr Andrew, Saskatoon, Saskatchewan, Canada,	1912
	Crothers, Dr Robert, Codrington Mansions, 139 Western Road, Brighton,	1913
160	Crow, Dr W. J., 108 Morningside Road,	1909
	Currie, Dr A. S., 20 Oxford Terrace, Hyde Park, London, W.,	1882
	Darling, Dr T. Brown, 13 Merchiston Place,	1884
	Darling, Dr William, 2 Warrender Park Terrace,	1900
	Davidson, Dr Samuel, Mansefield, Kelso,	1906
165	Davies, Dr Arnold, Menai Bridge, Anglesey,	1906
	Dendle, Dr Frank, Overton House, Spring Grove, Isleworth,	1892
	Dewar, Dr J. M., 24 Lauriston Place,	1910
	Dewar, Dr M., 24 Lauriston Place,	1891
	Dickie, Dr J. T., 37 Lauriston Place,	1906
170	Dickson, Dr D. Elliot, Hillcrest, Lochgelly,	1908
	Dickson, Dr George A., Invermae, South Queensferry,	1912
	Dickson, Dr George, 26 Merchiston Park,	1901
	Dobie, Dr D. Robertson, 7 James Square, Crieff,	1894
	Donald, Dr George, 46 Ferry Road, Leith,	1912
175	Drennan, Dr Matthew R., 35 Polwarth Gardens,	1910
	Dunbar, Dr H. J., 118 Cathedral Road, Cardiff,	1905
	Dyer, Dr Ethelbert W., Lustleigh, 132 Davenport Road, Durban, South Africa,	1906
	Easterbrook, Dr A. M., Arnprior, Gorebridge,	1895
	Easterbrook, Dr C. C., Crichton House, Dumfries,	1892
180	Eden, Dr T. Watts, 26 Queen Anne Street, Cavendish Square, London, W.,	1888
	Edington, Dr D. C., Birbeck House, Penrith,	1897
	Elder, Dr Edward A., 6 Torphichen Street,	1906
	Elder, Dr Eleanor, 4 John's Place, Leith,	1903
	Elder, Dr W. Nicol, 6 Torphichen Street,	1879
185	Elliott, Dr George Maxwell, Royal Maternity Hospital,	1912
	Evans, Dr Charles Hotham, 121 Harley Street, London, W.,	1912
	Evans, Dr O. F., 20 Princes Avenue, Liverpool,	1890
	Fairfax, Dr Norman P., Caddon View, Innerleithen,	1912
	Farie, Dr G. J., Strathallan, 143 Worple Road, Wimbledon, London, S.W.,	1903

		Date of Admission.
190	Farquharson, Dr J. D., 242 Westgate Road, Newcastle-on-Tyne,	1890
	Felkin, Dr R. W., 47 Bassett Road, North Kensington, London, W.,	1884
	Fergusson, Dr Samuel, Lylestone House, Alloa,	1912
	Finlay, Dr Thomas, 8 Comely Bank Road,	1912
	Finlay, Dr T. Y., 42 Comiston Drive,	1908
195	Fitzgerald, Dr Gordon, 183 High Street, Manchester,	1913
	Fitzwilliams, Dr Duncan C. L., 31 Grosvenor Street, Grosvenor Square, London, W.,	1904
	Fleming, Dr Andrew, St John's Road, Corstorphine,	1905
	Flett, Dr A. B., 15 Walker Street,	1903
	Fordyce, Dr William, 20 Charlotte Square,	1888
200	Forrest, Dr Stephen, 1 Magdala Crescent,	1910
	Fothergill, Dr W. Edward, 337 Oxford Road, Manchester,	1894
	Fowler, Dr W. Hope, 21 Walker Street,	1900
	Fox, Dr J. W., 18 Bernard Street, Southampton,	1887
	Fraser, Dr J. Hossack, Fernfield, Bridge of Allan,	1895
205	Fraser, Dr Nutting S., 205 Gower Street, St John's, Newfoundland,	1887
	Frost, Dr W. E., 36 Moray Place,	1900
	Garbutt, Dr W. J., Girthen House, Bournbrook Rd., Selly Pk., Birmingham,	1897
	Gardiner, Dr Frederick, 60 George Square,	1900
	Gardner, Dr L. P. M., 20 Pilrig Street,	1912
210	Gemmell, Dr J. E., 28 Rodney Street, Liverpool,	1885
	Gibbs, Dr J. H., 12 Coates Crescent,	1905
	Gibson, Dr Alexander, 14 Scotland Street,	1908
	Giffen, Dr J. T. M., 138 Boughton, Chester,	1892
	Giles, Dr A. B., 4 Palmerston Place,	1891
215	Gilmour, Dr T. F., Port Ellen, Islay,	1882
	Godfrey, Dr W. F., Carberry, Manitoba, Canada,	1906
	Godwin, Dr H. J., The Friary, Winchester,	1913
	Graham, Dr A., Curriebank, Currie,	1897
	Graham, Dr D. J., 2 Melville Crescent,	1895
220	Graham, Dr F. M., 16 Mayfield Gardens,	1894
	Graham, Dr R. Balfour, Leven Bank, Leven, Fife,	1893
	Grant, Dr A. G. M., The Manse, Stoer, Lochinver,	1914
	Gray, Dr J. A., 4 Wolseley Terrace,	1907
	Green, Dr John Ligertwood, 23 Minto Street,	1902
225	Gregory, Dr W. H., North Bar Without, Beverley, Yorks,	1893
	Greig, Dr F. J., Lt.-Col. R.A.M.C., 6 Gladstone Place, Stirling,	1906
	Gunn, Dr James T., Deanswood, Auchterarder,	1912
	Gunn, Dr J. A., 39 Morningside Drive,	1906
	Guthrie, Dr A. Cowan, 13 Coates Crescent,	1888
230	Guthrie, Dr D. T., 8 Strathearn Road,	1914
	Hamilton, Dr J. R., Elm House, Hawick,	1879
	Hamp, Dr J. Walton, Penn Road, Wolverhampton,	1886
	Harper, Dr R. A. J., 135 Abbey Road, Barrow-in-Furness,	1901
	Hartley, Dr J. N. Jackson, The Residency, Royal Infirmary, Edinburgh,	1914
235	Harvey, Dr Charles, Beckford Lodge, Sav-la-Mar, Jamaica, W.I.,	1889
	Harvey, Dr James, 7 Blenheim Place,	1891
	Havelock, Dr John G., Sunnyside, Montrose,	1888
	Hay, Dr Henry, 11 Great King Street,	1879
	Heard, Dr Richard, c/o Grindlay & Co., 54 Parliament Street, London, S.W.,	1912

		Date of Admission.
240	Hellier, Dr J. B., 27 Park Square, Leeds,	1904
	Helm, Dr John H., Clarence Cottage, Ratho,	1888
	Helme, Dr G. Edgar, Gloucester House, Rusholme, Manchester,	1895
	Hendrick, Dr A. C., 646 Spadina Avenue, Toronto,	1912
	Hewat, Dr A. Fergus, 3 Darnaway Street,	1912
245	Hewat, Dr A. Middleton, 2 West Road, Fulwood, Preston,	1907
	Hewetson, Dr J., Holmfield, Reigate,	1881
	Hindmarsh, Dr Edwin, Mozufferpore, Tirhoot State Railway, Bengal, India,	1895
	Hoggan, Dr Robert, Liberton Park, Liberton,	1894
	Honeyford, Dr John, 12 Warrender Park Terrace,	1912
250	Howard-Jones, Dr M. H., 1 Strathfillan Road,	1910
	Hughes, Dr Percy T., Barnsley Hall, Bromsgrove, Worcestershire,	1895
	Hume, Dr Wm. Maitland, 7 Atholl Crescent, Perth,	1911
	Hunter, Dr A. J. 17 Walker Street	1910
	Hunter, Dr J. H. Graham, Royal Maternity Hospital, Lauriston Place,	1914
255	Huskie, Dr David, Hamilton House, Moffat,	1912
	Hutchison, Dr Alice M., 204 Bruntsfield Place,	1905
	Inch, Dr T. Douglas, Stobsmills House, Gorebridge,	1914
	Inglis, Dr Elsie M., 8 Walker Street,	1901
	Jamieson, Dr Hugh, 1 Strathearn Road,	1889
260	Jamieson, Dr J. Boyd, 43 George Square,	1900
	Jardine, Dr F. E., Evelyn Cottage, Penicuik,	1910
	Jardine, Dr Robert, 20 Royal Crescent, Glasgow, W.,	1897
	Johnston, Dr G. Minto, 7 Wellington Place, Leith,	1912
	Johnston, Dr Robert B., Bishopyards, Penrith,	1903
265	Johnston, Dr R. J., 18 George Square,	1899
	Johnstone, Dr R. W., 10 Alva Street,	1903
	Keay, Dr J. W., 9 Granville Terrace,	1903
	Keir, Dr Ivan C., The Limes, Melksham, Wilts,	1903
	Kerr, Dr J. M. Munro, 7 Clairmont Gardens, Glasgow,	1894
270	Kerr, Dr J. Wishart, 151 Greenhead Street, Glasgow,	1901
	King, Dr J. K., The Glen Springs Sanitorium, Watkins, New York, U.S.A.,	1884
	Kirk, Dr Robert, Rowan Bank, Bathgate,	1887
	Kramer, Dr Hermann, 14 Leven Terrace,	1912
	Kynoch, Professor Campbell, 8 Airlie Place, Dundee,	1892
275	Lackie, Dr James Lamond, 7 Randolph Crescent,	1889
	Langwill, Dr Arch., Chalmers Hospital, Lauriston Place,	1913
	Langwill, Dr James, 4 Montebello, Joppa,	1911
	Langwill, Dr H. G., 4 Hermitage Place, Leith,	1891
	Lawrence, Dr H. Ruthven, Midwood, Palmyra Road, Newlands, Cape Colony,	1908
280	Lawrie, Dr Thomas Harcourt, St Clair, Polmont Station, Stirlingshire,	1912
	Ledger, Dr A. G. K., Westerton, Coppull, Lancashire,	1906
	Lee, Dr Herbert E., Coolabah, Belmore Street, Burwood, N.S.W., Australia,	1892
	Leebody, Dr H. A., Corstorphine,	1912
	Leslie, Dr W., 17 Mar Street, Alloa,	1912
285	Littlejohn, Professor Harvey, 11 Rutland Street,	1890
	Lochhead, Dr James, The Colonial Hospital, Gibraltar,	1904
	Lockhart, Dr F. A. L., 23 Mackay Street, Montreal, Canada,	1890
	Lorimer, Dr Duncan, 74 Bruntsfield Place,	1906
	Low, Dr R. Cranston, 7 Castle Terrace,	1902
290	Lyle, Dr R. P. Ranken, 11 Ellison Place, Newcastle-on-Tyne,	1901

		Date of Admission.
	MacArthur, Dr D. G., 14 Barkston Gardens, London, S.W.,	1903
	M'Cann, Dr F. J., 14 Wimpole Street, Cavendish Square, London, W.,	1896
	Macdonald, Dr A. G., Uig, Isle of Skye,	1903
	Macdonald, Dr Angus, Olive Lodge, 42 Polwarth Terrace,	1897
295	Macdonald, Dr John, Marathon House, Cupar-Fife,	1902
	Macdonald, Dr W. Fraser, 16 Buckingham Ter., Glasgow, W.,	1884
	Macdonald, Dr W. Fraser, 96 Polwarth Terrace,	1910
	M'Ewan, Dr Peter, 294 Barkerend Road, Bradford,	1905
	M'Ewen, Dr Alexander, Thornhill, Crieff,	1912
300	M'Gibbon, Dr John, 24 Melville Street,	1902
	MacGlashan, Dr Keith B., 2 Hartley Hill, Leeds,	1910
	MacGregor, Dr Alastair, 6 Russell Road, Kensington, London, W.,	1905
	MacGregor, Dr A. V., Durham House, West Hartlepool,	1895
	Macgregor, Dr G. S., 15 Buckingham Terrace, Glasgow, W.,	1888
305	M'Ilwraith, Dr Kennedy C., 30 Prince Arthur Avenue, Toronto, Canada,	1901
	M'Intosh, Dr A. Morrison, 4 E. Claremont Street,	1912
	Mackay, Dr Malcolm E., Paynton, Saskatchewan, Canada,	1912
	Mackay, Dr W. B., 23 Castlegate, Berwick-on-Tweed,	1899
	M'Kellar, Dr George, 20 Bowmont Street, Kelso,	1912
310	M'Kendrick, Dr Archd., 27 Chalmers Street,	1906
	Mackenzie, Dr T. C., Ruigh-Ard, Inverness,	1900
	M'Kerron, Dr R. Gordon, 1 Albyn Place, Aberdeen,	1896
	Mackie, Dr George, Boyd's Lodge, Malvern, Worcestershire,	1900
	Mackin, Dr Patrick, 132 Vizean Street, Wellington, New Zealand,	1895
315	Mackness, Dr G. Owen C., Fort Street House, Broughty-Ferry,	1887
	M'Laren, Dr John, 39 Lauriston Place,	1910
	M'Laren, Dr Robert, 10 Gilmore Place,	1912
	M'Larty, Dr Malcolm, 23 Abercromby Place,	1900
	Maclean, Dr Ewen J., 12 Park Place, Cardiff,	1902
320	Macleod, Dr J. A., Glencorse House, Peebles,	1907
	M'Morland, Dr John B., Kilneiss, Moniaive, Dumfriesshire,	1906
	M'Morland, Dr R. P., Moniaive, Dumfries,	1907
	MacRae, Dr John, Lynwood, Murrayfield,	1893
	MacVie, Dr S., Herbertknowe, Chirnside,	1881
325	M'Watt, Dr John, Morelands, Duns,	1879
	Macwatt, Dr R. C., Lt.-Colonel I.M.S., P.O. Box 110, Bombay, India,	1885
	Malcolmson, Dr Alexander M., Dalveen, Corstorphine,	1901
	Mallace, Dr A. C., Sandhaven House, Culross, Fife,	1908
	Marshall, Dr G. Balfour, 19 Sandyford Place, Glasgow,	1891
330	Marshall, Dr L. R. H. P., Hollybush, Peebles,	1912
	Martin, Dr Angus, 25 Northumberland Square, North Shields,	1906
	Martin, Dr Charles, Kobe, Japan,	1892
	Martin, Dr J. W., Charterhall, Newbridge, Dumfries,	1887
	Matheson, Dr A. A., 41 George Square,	1887
335	Matheson, Dr Angus, Duntulm, North Berwick,	1912
	Matheson, Dr R. Murdoch, 33 Buccleuch Place,	1897
	Mathewson, Dr G. D., 6 Castle Terrace,	1905
	Meikle, Dr J. Hally, 12 Midmar Gardens,	1906
	Menzies, Dr W. Menzies, 25 Castle Terrace,	1910
340	Michael, Dr Gustave, Bayham House, 135 West End Lane, West Hampstead, London, N.W.,	1885
	Millard, Dr W. W., Middlefield House, Leith Walk,	1884

		Date of Admission.
	Miller, Dr Alexander, 1 Royal Terrace, Crosshill, Glasgow,	1902
	Miller, Dr A. S., Rannoch House, Tranent,	1912
	Miller, Dr W. H., 51 Northumberland Street,	1886
345	Milligan, Dr Harley P., Hessle, East Yorks,	1908
	Milne, Dr W. M., 3 Minto Street,	1898
	Mitchell, Dr C. R. P., The Chase, Malvern,	1906
	Mitchell, Dr G. B., 1 Skinner Street, Whitby,	1893
	Montgomery, Dr John, The Highlands, Balsall Heath, Birmingham,	1895
350	Moorhouse, Dr J. Ernest, 6 Melville Terrace, Stirling,	1892
	More, Dr John, Rothwell, Kettering,	1908
	Morison, Dr Albert E., Mowbray Villa, Sunderland,	1888
	Morison, Dr Archibald Alexander, 33 Craiglea Drive,	1912
	Morris, Dr S. Glanville, Bryn-y-Delyn, Mardy, Glamorganshire,	1894
355	Moses, Dr O. St John, Medical College, 26 Park Street, Calcutta, India,	1900
	Mowat, Dr John, 5 Hope Park Terrace,	1884
	Mowat, Dr R. S., 40 Raeburn Place,	1910
	Muir, Dr Ernest, 17 Craiglea Drive,	1914
	Munro, Dr J. Ramsay, High Street, Spalding,	1901
360	Murray, Dr James, 51 Great King Street,	1879
	Napier, Dr A. D. Leith, 28 Angas Street, Adelaide, South Australia,	1878
	Nasmyth, Dr Alexander, Hillwood House, Penicuik,	1912
	Nicholson, Dr H. Oliphant, 20 Manor Place,	1900
	Ogden, Dr O. Watson, 38 Jesmond Road, Newcastle-on-Tyne,	1900
365	Ogilvy, Dr Stewart Grant, Fairmont, Fauldhouse,	1895
	Oliphant, Dr E. H. Lawrence, 23 Newton Place, Glasgow,	1885
	Olivier, Dr P. J., South African Union, 14 Buccleuch Place,	1911
	Orbell, Dr Ronald S., 12 Reed Street, Oamaru, New Zealand,	1907
	Orr, Dr John, Heather Lea, Clarendon Road, Eccles, Lancs.,	1887
370	Orr, Dr John, 6 Strathearn Road,	1912
	Orr, Dr T. S. A., Moray House, Crail,	1912
	Orr, Dr W. Basil, 13 Braid Road,	1889
	Patch, Dr C. Lodge, Renfrew District Asylum, Dykebar, Paisley,	1913
	Paterson, Dr G. Keppie, 19 Albany Street,	1882
375	Paterson, Dr G. W. Simla, 147 Bruntsfield Place,	1896
	Paterson, Dr William, 8 Hermitage Place, Leith,	1912
	Pearson, Dr C. Mowbray, 14 Manor Place,	1902
	Pearson, Dr John H. H., 12 Primrose Bank Road, Trinity,	1912
	Peddie, Dr H. Anderson, 24 Palmerston Place,	1882
380	Pell, Dr J. W., 45 Merchiston Crescent,	1914
	Perigal, Dr Arthur, New Barnet, Herts,	1887
	Petersen, Dr George R., Saskatoon, Saskatchewan, Canada,	1912
	Playfair, Dr John, 5 Melville Crescent,	1873
	Poole, Dr T. D., North Side House, Linthwaite, Huddersfield,	1890
385	Porritt, Dr E. E., Wanganui, New Zealand,	1897
	Porteous, Dr J. Lindsay, 87 North Broadway, Yonkers, New York,	1875
	Porter, Dr Frederick, 65 Morningside Road,	1904
	Prentice, Dr W. H., Hollybank, Eccles, Old Road, Pendleton, Manchester,	1901
	Price, Dr A. W. Gordon, 41 Minto Street,	1890
390	Price, Dr E. F. T., 13 Alva Street,	1890
	Primrose, Dr Alex., 100 College Street, Toronto, Canada,	1887
	Pringle, Dr J. Hogarth, 172 Bath Street, Glasgow,	1886

		Date of Admission.
	Proudfoot, Dr Frank Grégoire, 9 Chalfont Road, Oxford, .	1913
	Rabagliati, Dr A. H., c/o H. A. Jeffrey, Esq., Solicitor, 13 Cheapside, Bradford, Yorkshire, . . .	1903
395	Reid, Dr H. S., 3 Hampton Terrace, ...	1907
	Rendell, Dr Herbert R., P.O. Box 606, St John's, Newfoundland, ...	1882
	Ritchie, Dr George B., 9 Brunswick Street, . . .	1912
	Ritchie, Dr James, 22 Charlotte Square, . . .	1880
	Ritchie, Dr T. C., Glasgow Maternity Hospital, . .	1911
400	Robarts, Dr Henry H., 1 Wemyss Place, Haddington, .	1904
	Roberton, Dr Ernest, Remnack Road, Auckland, New Zealand,	1887
	Roberts, Dr D. Lloyd, 11 St John Street, Manchester, .	1906
	Robertson, Dr George, Braehead, Viewfield Place, Dunfermline, ...	1901
	Robertson, Dr Robert, 26 Royal Circus, . . .	1897
405	Robertson, Dr R. Macdonald, 53 Bruntsfield Place, . .	1912
	Robertson, Dr W. B., St Anne's, 101 Thurlow Park Road, Dulwich, London, S.E., ...	1870
	Robinson, Dr H. Shapter, Talfourd House, 78 Peckham Road, Camberwell, London, S.E., ...	1890
	Ronaldson, Dr R. Miller, 9 Melville Crescent, . .	1912
	Rosa, Dr Albert F., 28 Pitt Street, ...	1893
410	Rosair, Dr H. Basil, c/o Thomas Cook & Son, Ludgate Circus, London, ...	1912
	Ross, Dr A. Aitken, 8 Newington Road, . . .	1910
	Ross, Dr T. W. E., 31 Morningside Drive, . . .	1904
	Russell, Dr W., 3 Walker Street, ...	1890
	Saleeby, Dr C. W., 10 Camden Mansions, The Mall, London, W., ...	1902
415	Sandstein, Dr Alfred C., 21 Latimer Square, Christchurch, New Zealand, ...	1898
	Saunders, Dr F. A., Hill Street, Grahamstown, Cape Colony, South Africa, ...	1885
	Scannell, Dr F. A., 11 Eyre Place, Edinburgh, and Ma Waro, Timaru, New Zealand, ...	1912
	Sclater, Dr W. O., Ipoh, Perak, F.M.S., . . .	1906
	Scott, Dr Alexander, The Firs, Broxburn, . . .	1906
420	Scott, Dr T. R., 13 High Street, Musselburgh, . .	1880
	Secord, Dr E. R., 112 Market Street, Brantford, Ontario, Canada, ...	1902
	Selkirk, Dr W. J. Burns, 3 Bayswell Road, Dunbar, . .	1912
	Shannon, Dr David, 270 Bath Street, Glasgow, . . .	1910
	Shaw, Dr C. J., Argyle and Bute Asylum, Lochgilphead, .	1903
425	Shearer, Dr Alfred, The Bank, Newtown, N. Wales, . .	1898
	Sibbald, Dr T. R. S., Stanley, Perthshire, . . .	1912
	Silberbauer, Dr Stanley F., Provincial Chambers, 14 Keerom Street, Cape Town, South Africa, ...	1912
	Simpson, Dr Archibald, 16 Swan Hill, Shrewsbury, . .	1906
	Simpson, Dr F. D., 9 Hampton Terrace, . . .	1900
430	Slight, Dr J. D., 163 London Road, Leicester, . .	1898
	Sloan, Dr Allen T., 22 Abercromby Place, ...	1893
	Sloan, Dr S., 5 Somerset Place, Sauchiehall Street, West, Glasgow, ...	1887
	Smart, Dr David, 74 Hartington Road, Sefton Park, Liverpool,	1882
	Smith, Dr G. H. Walton, Pendower, Oxford St., Paddington, Sydney, Australia, ...	1892
435	Smith, Dr Gains T., 71 Church Street, Moncton, New Brunswick, Canada, ...	1891
	Smith, Dr John, Bryce Hall, Kirkcaldy, . . .	1885

		Date of Admission.
	Smith, Dr W. Ramsay, Central Board of Health, Adelaide, South Australia,	1892
	Smith, Dr W. Torrance, 70 Woodlands Road, Aigburth, Liverpool,	1906
	Sneddon, Dr William, Logan Bank, Cupar-Fife,	1888
440	Somerville, Dr C., Tuling, Dalkeith,	1902
	Somerville, Dr James W., The Grange, Galashiels,	1902
	Spence, Dr John W. L., 22 Pitt Street,	1912
	Stark, Dr J. Nigel, 4 Newton Place, Charing Cross, Glasgow,	1908
	Stephen, Dr W. A., Loftus-in-Cleveland, Yorkshire,	1894
445	Stephenson, Professor Wm., 3 Rubislaw Terrace, Aberdeen,	1861
	Steuart, Dr Murray B., Oakley, Kirkcudbright,	1912
	Stevens, Dr John, 78 Polwarth Terrace,	1894
	Stewart, Dr A. D., 8 Brougham Place,	1906
	Stewart, Dr A. M., 146 Ferry Road, Leith,	1913
450	Stewart, Dr J. S., 173 Bruntsfield Place,	1884
	Stewart, Dr Peter, The Lindens, Uphall,	1912
	Stewart, Dr R., 25 George Square,	1885
	Stewart-Sandeman, Dr Laura, 22 Waverley Place, Aberdeen,	1912
	Stirling, Dr R., 4 Atholl Place, Perth,	1892
455	Strain, Dr Arthur C., 3 Clifton Avenue, West Hartlepool,	1904
	Stumbles, Dr H. M., 5 Wilbury Gardens, Hove, Sussex,	1903
	Sturrock, Dr J. F., Arima, Broughty-Ferry,	1887
	Tannahill, Dr W. B., 40 Margaret Street, Greenock,	1907
	Taylor, Dr David R., St Helen's, Ayton,	1896
460	Taylor, Dr William, 12 Melville Street,	1868
	Taylor, Dr W. Macrae, 8 Melville Street,	1895
	Teacher, Dr Charles C., Craigend, North Berwick,	1887
	Temple, Dr G. H., Ailanthus, Weston-super-Mare,	1888
	Tennant, Dr John, 89 Frodingham Road, Scunthorpe, Doncaster,	1902
465	Thatcher, Dr C. H., 8 Melville Crescent,	1877
	Thatcher, Dr L. H. F., 8 Melville Crescent,	1910
	Thin, Dr Robert, 25 Abercromby Place,	1891
	Thomas, Dr G. Crewdson, 10 Chester Square, London, S.W.,	1899
	Thompson, Dr F. E., 20 Park Avenue, Montreal, Canada,	1902
470	Thompson, Dr Herbert P., Beech Lodge, 2 Esplanade, Harrogate,	1906
	Thompson, Dr James L., Castlemaine, Victoria, Australia,	1894
	Thomson, Dr A. C., 11 Eyre Place, Edinburgh, and 4 Burwood Avenue, Newington, Dunedin, New Zealand,	1912
	Thomson, Dr A. D. R., 19 Bridge Street, Musselburgh,	1877
	Thomson, Dr John, 14 Coates Crescent,	1887
475	Thomson, Dr T. J., 3 Greenhill Park,	1902
	Thyne, Dr T. J., 16 Randolph Crescent,	1887
	Tod, Dr John, 67 Ferry Road, Leith,	1892
	Tristan, Dr R. J., 28 Carolgate, Retford, Notts,	1887
	Tuke, Dr A. L. S., 12 Comely Park, Dunfermline,	1911
480	Turnbull, Dr A. E., Coxwell Road, Farringdon, Berks,	1909
	Underhill, Dr T. Edgar, Dunedin, Barnt Green, North Birmingham,	1879
	Vassie, Dr Alexander H., 98 Priory Road, West Hampstead, London, N.W.,	1891
	Veitch, Dr Alexander, 12 Gilmore Place,	1912
	Venters, Dr Isabel, 6 Archibald Place,	1907
485	Vickery, Dr W. H., 1 Trewartha Park, Weston-super-Mare,	1892
	Walker, Dr Arthur S., School Board Offices, Leith,	1906
	Walker, Dr Ernest A., c/o Thomas Cook & Sons, Ludgate Circus, London,	1912

		Date of Admission.
	Wallace, Dr Abraham, 39 Harley Street, London, W.,	1879
	Wallace, Dr R. W. L., Mesopotamia Irrigation Service, Sir John Jackson, Ltd., Bagdad, Turkey-in-Asia,	1905
490	Watson, Dr Alexander E., East Wemyss, Fife,	1912
	Watson, Dr Anne Mercer, 22 Waverley Place, Aberdeen,	1912
	Watson, Dr B. P., University, Toronto,	1902
	Watson, Dr R. H., Trereiffe, Roche, R.S.O., Cornwall,	1896
	Watt, Dr Robert Honey, Esk Villa, Langholm, Dumfries,	1912
495	Waugh, Dr John, 36 Finsbury Pavement, London, E.C.,	1881
	Webb, Dr H. W., Royal Infirmary,	1913
	Webster, Prof. J. C., Rush Medical College, 1746 W. Harrison Street, Chicago, U.S.A.,	1889
	Welsh, Dr Tom, 8 Chamberlain Road,	1912
	Wemyss, Dr H. L. Watson, 7 Coates Gardens,	1910
500	White, Dr A. L., 145 Manchester Road, Rochdale, near Manchester,	1903
	Wilkie, Dr James, Selville House, Portobello,	1897
	Wilkinson, Dr George, 3 Dingle Hill, Liverpool, S.,	1889
	Will, Dr J. C. Ogilvie, 17 Bon-Accord Square, Aberdeen,	1867
	Willcox, Dr F. Mayes, 8 Strathearn Road,	1902
505	Wilson, Dr James, 53 Inverleith Row,	1889
	Wise, Dr Robert, 37 Vartry Road, Stamford Hill, London, N.,	1890
	Wood, Dr A. Murray, 180 Ferry Road, Leith,	1912
	Wood, Dr Thomas, 182 Ferry Road,	1886
	Woodside, Dr J. T., 13 Meadow Street, Aberkenfig, R.S.O., Glamorganshire,	1898
510	Yoe, Dr Richard T., 415 W. Chestnut Street, Louisville, Kentucky, U.S.A.,	1891
511	Young, Dr James, 2 Randolph Place,	1907

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TRANSACTIONS

OF THE

EDINBURGH OBSTETRICAL SOCIETY,

FOR SESSION LXXV., 1913–1914.

MEETING I.—NOVEMBER 12, 1913.

Dr J. HAIG FERGUSON, *President, in the Chair.*

I. *The Treasurer* submitted his Annual Statement.

INCOME.

Balance from Session 1911-1912,					£581	8 7
Arrears,	£17	4	11			
Bank Interest on Deposit Receipts,	2	11	9			
Interest on Consols,	5	19	8			
Interest on N.B.R. Debenture Stock,	8	9	6			
Entrance Fees from 20 new Ordinary Fellows,	21	0	0			
Contributions for current year from 413 Ordinary Fellows,	103	5	0			
Composition for Life-Membership from 5 Ordinary Fellows,	26	5	0			
Transactions sold,	6	3	0			
Refund by Dr M'Cann for Blocks,	14	6	6			
Refund by Dr Haig Ferguson for Blocks,	2	12	6			
					207	17 10
					£789	6 5

EXPENDITURE.

Corporation Duty,	£0	15	3			
Income Tax,	0	2	4			
Shorthand Reporter,	8	8	0			
Commission to Collector,	1	0	9			
Door-keeper's Salary,	1	12	0			
Oliver & Boyd's Account for Vol. XXXVIII. of Society's *Transactions*,	142	15	9			
Oliver & Boyd's Account for Printing Billets, Postages, etc.,	27	5	0			
Rent of Rooms,	4	10	0			
Waterston & Sons' Account,	4	5	0			
A. Frazer's Account,	0	12	0			
G. Yeoman, use of Lantern and Operator,	1	10	0			
Treasurer, Secretary, and Editor,	0	17	6			
Professor Jardine for Blocks,	2	15	0			
					£196	8 7
Society's share of cost of Epidiascope,					44	0 2
Balance to New Account,					548	17 8
Made up as follows:—						
* Invested Funds,	£505	6	6			
Deposit Receipt,	35	19	10			
Due by Bank,	20	2	4			
	£561	8	8			
Less Contributions for Sessions in advance.	12	11	0			
	£548	17	8		£789	6 5

* The Invested Funds are—£250, 10s. 6d. in 2½ per cent. Consols, and £254, 16s. in 3 per cent. N.B.R. Debenture Stock.

Dr Borrowman and *Dr Graham* were appointed auditors, and found the accounts correct.

II. *The Secretary* (Dr Angus Macdonald) gave notice of motion, recommended by Council:—

> That the second sentence of Law XVIII. of the Society's Laws, which reads at present—"Fellows who are three years in arrear are liable to have their names deleted from the roll of the Society," *shall be altered to read*—"Fellows who are three years in arrear shall cease to be Fellows of the Society."

III. The Society then proceeded to the election of Office-bearers, and the President announced that the following were duly elected:—*President*, Sir Halliday Croom; *Vice-Presidents*, (*Senior*) Dr G. Barbour Simpson, (*Junior*) Dr Keppie Paterson; *Treasurer*, Dr John M'Gibbon; *Secretaries*, (*Senior*) Mr E. Scott Carmichael, (*Junior*) Dr R. W. Johnstone; *Librarian*, Dr Lamond Lackie; *Editor of Transactions*, Dr James Young; *Members of Council*, Professor M'Kerron, Dr J. W. Keay, Dr Oliphant Nicholson, Dr H. M. Church, Dr Haig Ferguson, Dr Angus Macdonald, Dr Berry Hart, Dr H. S. Davidson.

Sir Halliday Croom proposed a vote of thanks to the retiring Secretary (Dr Angus Macdonald), and said he knew of no more important work than that of the Secretary; the whole success of the Society to a great extent depends on him, and he thought of all the secretaries they had had, there had been none better than Dr Macdonald.

The President conveyed the cordial thanks of the Society to Dr Macdonald for his indefatigable work in connection with it.

IV. The following gentlemen were elected Ordinary Fellows of the Society:—T. W. J. Childs, M.B., Ch.B., Royal Maternity Hospital, Edinburgh; Thomas William Clarke,

M.B., Ch.B., The Royal Infirmary, Sheffield; H. J. Godwin, M.B., F.R.C.S.Ed., The Friary, Winchester; Archibald Langwill, M.B., Ch.B., Chalmers Hospital, Edinburgh; Frank Grégoire Proudfoot, M.D., 9 Chalfont Road, Oxford; A. M. Stewart, M.B., Ch.B., 146 Ferry Road, Leith; J. D. Grahame Stewart, M.B., Ch.B., Royal Maternity Hospital, Edinburgh.

V. VALEDICTORY PRESIDENTIAL ADDRESS.

By James Haig Ferguson, M.D., F.R.C.P., F.R.C.S.

When two years ago you did me the great honour of electing me to be your President, I accepted the high compliment and the responsibilities with considerable diffidence and hesitation, confiding more in your proved kindness and consideration to make things possible for me, than in my own ability to act as your representative and mouthpiece for the time being. In this forecast I have not been disappointed, and my expectations have been more than fully realised, despite my shortcomings, in the kindness, courtesy, and help I have invariably received from the Council and from the Society at large—a support without which it would have been impossible for me to have attempted to maintain the dignity of the Chair which I am about to demit. This kindness on your part, moreover, has made my term of office one of the pleasantest of my life, and one which has not been marred by one single note of discord—an experience which I venture to think is not always vouchsafed to chairmen,—and I am correspondingly grateful to you for having made this possible in my case.

No President could have had a more loyal or enthusiastic Council than I have had, and though it is invidious to single out names, yet I cannot but specially mention our indefatigable Secretaries, who have worked so zealously, and to whom the success of our meetings must be greatly attributed. To our

selfish regret we parted with our junior Secretary, Dr B. P. Watson, a year ago, but we cannot grudge him to our fellow-countrymen on the other side of the Atlantic. Our good wishes follow him in the important chair he has been called to occupy in the University of Toronto, where we are sure he will nobly uphold the great traditions of Edinburgh. We have been fortunate in securing the services of an equally energetic Secretary in the person of Mr Scott Carmichael.

Our Society is about to enter into the seventy-fifth year of its existence, and many of our younger Fellows will have the privilege of seeing the centenary. This, I doubt not, will be celebrated with the dignity and *éclat* equal to that which I so well remember characterised its jubilee, under the Presidency of my honoured friend the late Dr Underhill. Time will show if Dr Ballantyne's foreshadowing of the centenary will be an accurate one or no. It would not be inappropriate if *he* were in the Chair on the occasion, when he would be able to contrast prophecy with facts, and perhaps at the same time demonstrate that truth is sometimes more wonderful than fiction, and reality than romance.

Gentlemen, if, unfortunately, we as individuals are growing older, and some of us less energetic, yet our Society is renewing her youth, and gaining in vigour and in numbers as the years roll on.

I welcome you to-night to our first meeting in our new hall, which I believe opens up a new era in our history. We have combined with our sister Society (the Medico-Chirurgical Society) in securing, through the courtesy of Sir Robert Philip, this hall as our common meeting place, and in the acquisition (along with the authorities of the Royal Victoria Dispensary) of a first-class epidiascope, which latter is now an essential for modern scientific meetings. The mutual advantages of this arrangement, not only to the Societies themselves, but to the medical body corporate, are obvious—"ἐν τῳ δεσμῳ ἡ ἰσχυς."

We are entering on this new era with fresh and growing vitality. In these days of specialism there is an increasing tendency to drift apart, and therefore a common meeting-ground is a healthy antidote to such a state of matters. The more societies have in common, and the more they see of each others' work (as in the case of individuals), the more as a rule will they respect each other, and the less chance is there of misunderstandings being likely to arise. I hope this co-operation and unity may pave the way to occasional meetings in common, to discuss subjects of mutual interest which can with advantage be looked at from different points of view, thus giving the discussions perhaps a wider interest and importance. No better augury of this could be possible than Major Leonard Darwin's address last month, in which he so happily inaugurated our new venture.

Our Librarian informs me that we have met in St Andrew Square for forty-eight years, except for a short break in 1903-1904, when the St Andrew's Hall was undergoing renovation, when we removed temporarily to 18 George Street. Before that the Society met in the New Town Dispensary (where it originated), subsequently in 13 Queen Street, and then in the houses of the office-bearers in rotation, returning again to the New Town Dispensary in 1847, and moving to 5 St Andrew Square in 1865. In June 1880 there was a proposal that we should join with the other Edinburgh Scientific Societies and meet in a common hall. Drs Craig and Berry Hart were chosen as our representatives to attend the meeting and report to the Society. Under the date 30th June 1880 it is minuted:— "Report received, and it was agreed not to join in the scheme."

Let us hope that in the years to come our successors in this hall will look back as unashamedly as we can do to-night to those who have gone before us; and on this historic spot formerly owned by Mr Richard Lawson of the High Riggs (who is said to have heard at the Mercat Cross the dread summons of Pluto

before Flodden), may we and those who follow us feel the inspiring presence of our goddess Lucina and her heaven-born art.

Reference to our two last volumes of *Transactions* shows an array of practical and scientific contributions which reflects infinite credit on the activity of our Fellows, and which would do honour to any society, in their varied interest and importance.

We have had twenty-three communications dealing with Obstetrics, thirteen with Gynæcology, and one with new-born children; besides large numbers of interesting and instructive speciments exhibited, and some beautiful lantern demonstrations.

To mention only a few:—Dr M'Cann's admirably illustrated paper on Abdominal Hysterectomy for Cancer; Sir Halliday Croom's, Professor Jardine's, Dr Livingston's and Dr Haultain's, papers on Eclampsia; Mr George Chiene's lucid demonstration of Ovarian Pregnancy; Sir Halliday Croom's case of "Cerebral Tumour in Pregnancy simulating Eclampsia"; Dr Berry Hart's papers on the "Free-Martin" and on the "Biometric curves indicating the interval between Insemination and Impregnation in Mammals"; and the discussion on our old friend the Retraction Ring, introduced in a masterly manner by Dr Freeland Barbour, are amongst those which occur to one as of outstanding importance.

The discussions have been particularly well maintained, and on two occasions we were favoured by the presence of distinguished guests, who added greatly to the value of these discussions in the domain of Veterinary Medicine and Pathology respectively.

While, of course, welcoming papers of scientific and practical interest from the teacher and specialist, we are particularly anxious to encourage short contributions and records from the general practitioner, on obstetric cases. There is a feeling of diffidence on the part of the practitioner to come

forward, in case of severe or adverse criticism. I can assure them that they need have no fear of such treatment at the hands of the Society. Those of us who are teachers have got an immense amount to learn from the practitioner, and the practitioner may possibly learn something from the specialist What we want is a free and fearless exchange of views and opinions, a record from the vast fields of experience of the general practitioner, in whose hands the chief obstetric practice of the country is. Teachers are sometimes apt to forget the special difficulties of the practitioner, and it would certainly be to our mutual advantage to have them from time to time impressed upon us.

In passing, it may be interesting to glance for one moment at the effect the National Insurance Act has had on the teaching of obstetrics and on maternity hospitals in Scotland.

You may possibly remember that in my address last year I ventured to forecast the difficulties which I feared might follow the working of the Act from a maternity point of view, if the details were to be carried out according to the literal interpretation of the measure. Fortunately the large powers possessed by the Commissioners and the influence of the General Medical Council, in conjunction with an amending Act, have enabled wise counsels to prevail, and many difficulties have now been removed.

The statistics which I now present of the Scottish maternity hospitals show, however, by the marked decrease of cases in every instance, that some readjustment is still necessary before the pendulum swings back again to a better state of equilibrium, as I think it will certainly do.

In Glasgow, from 1st January to 31st August of this year, there is a decrease in indoor and outdoor cases of 717.

In Aberdeen, for the same period for all cases, a decrease of 145.

In Edinburgh, a decrease of 381.

In Dundee the calculation is begun each year on 15th May; for the year 1912-1913, therefore, the figures include January to May 1913, and they show a decrease of 121 for that period as compared to 1911-1912.

Meantime, therefore, our field of teaching is undoubtedly restricted, our output of nurses is consequently less, and our difficulty in meeting even the bare obstetric necessities of the medical student is correspondingly increased.

It is impossible yet to say how the women themselves will benefit in the long run, both immediately and remotely.

Two papers on somewhat similar lines (both of which will repay perusal) have recently appeared, dealing with the question of obstetric teaching and the improvement of the standard thereof.

One is from America, from our Honorary Fellow, Professor Whitridge Williams, who deplores the fact that in America "we have as yet failed to train practitioners competent to meet the emergencies of obstetrical practice." This is a serious indictment, but entirely justified by the statistics he produces, where the general practitioner in some parts of America is shown to have a higher mortality in his obstetric practice than the midwife, owing probably to the fact that the imperfectly trained practitioner does not recognise his own limitations, with the result that he attempts what he really is not competent to deal with, often with deplorable consequences.

Professor Williams contends that practical midwifery must be regarded as a branch of surgery, and that the only feasible method by which assistants can obtain the necessary facility in surgical methods and technique is by the closest co-operation with the gynæcological department, or preferably by the union of the two departments.

In this opinion I entirely agree, and I venture to think that one cannot overestimate the benefits *to* midwifery which accrue

from its close association with gynæcology, benefits which in my opinion far outweigh the more usually recognised advantages of a knowledge of obstetrics to the gynæcological surgeon. To this end Professor Williams urges the extension of obstetric charities—free hospitals and out-patient services for the poor, and proper semi-charity hospital accommodations for those in moderate circumstances, keeping in remembrance the fact, as recently stated so admirably by Mr Arthur Balfour, that "to look upon a hospital merely as a place where those who were sick or hurt went for healing was a very narrow view, because, unless it was also a place in which the future leaders of the medical profession were to be trained, its usefulness would die out with the present generation."

Whitridge Williams further advocates the gradual abolition of midwives in our large cities and their replacement by obstetric charities. He regards attempts at improving midwives as an "almost impossible task," and suggests the greater development of visiting obstetric nurses, very much on the lines of our own Jubilee Nurses.

I fear, however, midwives are a necessity, especially in rural districts, and moreover their continuance is insisted on in our own Insurance Act, but I strongly hold that *if they are efficiently supervised*, they really essentially conform to Whitridge Williams' definition of visiting Obstetric Nurses.

It is this proper supervision that we are so anxious to obtain in a Scottish Midwives Bill. Most fervently do we hope that such a measure may soon be enacted. It is a necessary antidote, so far as the welfare of the women and their infants is concerned, to the maternity provisions of the Insurance Act, and no opportunity should be lost in urging upon the Government and upon our Members of Parliament the pressing need of such a measure, and the reasons therefor.

Another important point referred to in this address is the education of the American laity to the fact that "poorly

trained doctors are dangerous, that most of the ills of women result from poor obstetrics, and that poor women in fairly well-conducted free hospitals usually receive better care than well-to-do women in their own homes." The British laity would also be none the worse of a measure of information in this respect. It is amazing the ignorance of even otherwise well-informed people concerning this.

Only the other day I was asked in open court by a leading K.C., "Do you mean to tell the jury that it is better for a poor girl (in this case an unmarried girl) to go for her confinement to a maternity hospital, than to be confined in a comfortable home (which in her case meant a small cottage), surrounded by a fond father, a doting mother, and loving sisters?" He said, on my assuring him that such was the case, and that it was the general trend of modern medical opinion, that this was a strange and extraordinary statement to make. I may add, however, that the intelligent jury did not share his opinion.

The second writer I have referred to is Dr Victor Bonney, of London, who pleads for the necessity of recognising midwifery as a branch of surgery, and for the conduct of all labours in a true surgical environment. He also urges the importance of educating the public to the importance of labour being properly conducted under the principles of true surgical asepsis. His paper is well worth attention.

These ideals are what we should aim at, and each of us can do something, however small, in helping to bring them about. But,

> "Let no man think that sudden in a minute
> All is accomplished, and the work is done;
> Though with thine earliest dawn thou should'st begin it,
> Scarce was it ended in thy setting sun."

It is my sad duty as your retiring President to present to you a short notice of those Fellows who have passed away

during my term of office, and whose loss we are now called on to deplore. Their work in this world is already sealed by the past, whereas yours (at least that of the younger Fellows) is still rich in boundless possibilities.

Among our Honorary Fellows we miss the name of *George Granville Bantock*, who died in London last January, at the age of 76. He was elected an Honorary Fellow in 1897.

A graduate of Edinburgh University, he settled in London in 1865, and soon made a name for himself there, by his manual dexterity, especially in plastic operations on the perinæum and vagina. On Sir Spencer Wells' retiring from the Samaritan Hospital in 1878, he became a full surgeon there, and remained on the active staff till 1896. He distinguished himself greatly by his success as an ovariotomist and in advancing the surgery of uterine fibromyomata. He was one of the original founders of the British Gynæcological Society, and held the office of President in 1887.

Absolute cleanliness was Bantock's great aim, and he was recognised as one of the cleanest operators in London. His results were extraordinarily good, and were greatly due to his wonderful dexterity and scrupulous care. Many of us will remember the illuminating address he gave us here in 1904, "In defence of the Pessary." He wrote largely in the medical journals. His best known publication is probably the "Treatment of Ruptured Perinæum."

Professor Henry J. Garrigues also died this year. He was elected an Honorary Fellow in 1902.

Born in Copenhagen in 1831, he graduated there, and settled in New York in 1875. He become Obstetric Surgeon to the Maternity Hospital, and Professor of Gynæcology and Obstetrics in the School of Clinical Medicine. He introduced aseptic obstetrics in America in 1883.

He was the author of two highly popular text-books on Diseases of Women and on Obstetrics respectively, which went

through several editions, besides many other publications, and he has left his mark in the annals of his time.

Professor Paul Segond, of Paris (an Honorary Fellow since 1903), likewise passed away this year while yet in his full vigour.

His surgical scientific work was considerable, but his chief interest was in Gynæcology. He was an ardent advocate of the vaginal route for gynæcological operations, and his great operative skill in this direction tempted him sometimes to extend the scope of these operations a little too far. He did yeoman service, however, in calling attention to what might be done in this direction. He wrote largely on abdominal surgery, and won the Goddard Prize in 1880. He was an Honorary F.R.C.S.E., 1905.

Professor Sir William Japp Sinclair (an Honorary Fellow since 1905) died last year, at the age of 66. A graduate of Aberdeen, he spent all his professional life in Manchester, where in 1888 he became Professor of Obstetrics and Gynæcology in the Victoria University.

He was a strong, independent man, of varied interests and of very decided opinions. He was a forcible writer, and his monograph on Gonorrhœa and his admirable life of Semmelweis testify to his powers.

He was a pioneer in vaginal hysterectomy, and a strong supporter of para-vaginal section in preference to abdominal operations for uterine cancer. His was the brain which conceived the *Journal of Obstetrics and Gynæcology of the British Empire*, and it was mainly through his exertions that the journal was launched. He was also an original member of the Central Midwives Board of England, and showed great interest and activity in framing the Act and working out all its complicated details.

Failing health unfortunately curbed his energies for some two years before his death.

Among our Corresponding Fellows we mourn the loss of

Arthur Bentley—a man of great ability and energy, of a most lovable nature, and of whom it could be truly said that whatever he did he did well.

Professor James Chalmers Cameron, of M'Gill College, is another notable figure whose death has to be recorded. He had been an Ordinary Fellow since 1888. He did good work during his twenty-five years' occupancy of the Montreal Chair of Obstetrics, and he was an extremely popular man in Canada.

In our own immediate circle we have lost five of our Ordinary Fellows, namely—*Dr David Menzies, Dr Thomas Proudfoot, Dr David George Davidson, Dr John MacMillan*, and *Dr A. Minto M'Donald.*

They were all personal friends of our own—good men and true, whose presence we can recall from time to time at our meetings.

Peace be to their memory.

Although he retired from the Society some time ago, the name of *Dr John Linton* for years figured as one of our most respected Fellows, and the announcement of his recent death at the age of 75 was a source of sincere regret to his many friends and his former comrades here. As an assistant of Dr Matthews Duncan, and afterwards for eleven years Physician to the Royal Hospital for Sick Children, he gained an experience in midwifery and children's diseases which led to his acquiring an enormous practice, especially in these departments. During his forty-three years of practice in Edinburgh he brought 10,000 children into the world, and in one year is said to have attended no less than 460 labours!

He was a man greatly beloved, who served his day and generation well, and whose memory will ever be held in the highest regard.

In an address of this kind I cannot but add one word in

reference to that great and notable Seventeenth International Medical Congress which met in London in August last, and which I venture to think will stand out as one of the most remarkable medical meetings which have ever assembled, both in the importance of its work and in the influence and stimulus for good it will undoubtedly exert among all the nations of the world. Never before has the medical profession demonstrated its wonderful power and possibilities in such a striking manner, and this year will be a memorable one, if only for the extraordinary success of the London meeting, and its far-reaching potentialities and results, scientifically and socially, for the good of mankind.

In this world, where familiarity and mystery are so strangely entwined (and nowhere more than in the domain of Obstetrics) we are still groping in semi-darkness.

We *do* know what the Hebrew Prophet thought no man would *ever* know—"how the bones do grow in the womb of her that is with child"; but children are still what they were in the time of the prophet, and life is still a mystery—will it ever remain such?

Some of you will remember the well-known old Dutch picture, showing a mother coming with her daughter to a physician, who is investigating a flask of the daughter's urine for the presence of "Kiestine," so as to determine the question of pregnancy. It has provoked many a smile in modern days. But lo! Abderhalden has appeared among us with his "Ninhydrin," by which he claims to diagnose or negative the existence of pregnancy biologically, without reference to the ordinary physical signs or subjective phenomena. If further experience should confirm his observations the diagnosis of pregnancy can be reduced to an exact science, which will be an enormous stride, and will at once remove all dubiety and often great anxiety from the minds of doctor and patient alike.

I must, however, now bring my remarks to a conclusion, and I cannot more suitably do so than by a quotation from our new Poet Laureate—the first medical man who has ever adorned this high position—who so beautifully and aptly interprets the position of things medical at the present time:—

"And yet in all mankind's disappointed history, now first
Have his scouts pushed surely within his foul enemies' lines,
And his sharpshooters descried their insidious foe,
Those swarming parasites, that barely within the detection
Of manifold searchlight, have bred, swimming unsuspected
Thro' man's brain and limbs, slaying with loathly pollution
His beauty's children, his sweet scions of affection,
In fev'rous torment and tears, his home desolating
Of their fair innocence, breaking his proud passionate heart,
And his kindly belief in God's good justice arraigning.
. . . . . .
Grant us an hundred years, and man shall hold in abeyance
These foul distempers, and with this world's benefactors
Shall Pasteur obtain the reward of saintly devotion
His Crown heroic, who fought not destiny in vain."

And again, in more general terms:—

"O youth, whose hope is high,
Who dost to Truth aspire,
Whether thou live or die,
O look not back nor tire.
. . . . . .
If thou canst Death defy,
If thy Faith is entire,
Press onward, for thine eye
Shall see thy heart's desire."

And so I bid you farewell, not, I trust, as an individual, but as your President, and I confidently hand over my duties and privileges to my successor—my teacher and friend, Professor Sir Halliday Croom, whom, in your name as well as in my own, I cordially welcome to the Chair, which he has already on previous occasions so worthily filled and adorned.

References.

J. W. Ballantyne.—"Inaugural Address on the Future of Obstetrics," 14th November 1906, *Transactions Edin. Obst. Soc.*, vol. xxxii.

Professor Whitridge Williams.—"Medical Education and the Midwife Problem in the United States," *The Journal of the American Medical Association*, 6th January 1912, vol. lviii.

Victor Bonney.—"The necessity of recognising Midwifery as a branch of Surgery," *Brit. Med. Journ.*, 15th March 1913, p. 552.

Sir Halliday Croom, on taking the Chair, thanked the Society most cordially for the high honour they had done him in electing him to the Chair for the third time in his life. He realised the responsibility, and knew what it meant to take up the duties which Dr Haig Ferguson had so thoroughly fulfilled. He again welcomed the members to the new hall, and although he felt some regret at leaving the old hall in St Andrew Square, hoped they would have as distinguished a career in the new as in the old.

Dr Berry Hart proposed a very hearty vote of thanks to the retiring President. When Dr Haig Ferguson was elected to the Chair, all those who knew him were aware he would carry out the work in a most distinguished manner, and the way in which he had conducted himself, the specimens he had shown, and the very excellent papers he had given, especially the one on Hydatid Mole, had more than fulfilled their anticipations.

VI. A CASE OF CLOACAL VESICAL EXSTROPHY IN THE FEMALE.

By A. A. Morison, M.B., Ch.B. Ed. (Communicated by Dr T. B. Johnston.)

Through the kindness of Sir Halliday Croom I have been permitted to place on record a case of cloacal vesical exstrophy in the female, which occurred in the out-patient practice of the Edinburgh Royal Maternity Hospital.

The child was born on 4th December 1910.

The maternal history is of interest as regards the previous confinements.

Mrs K., æt. 32, very healthy till time of her marriage, which took place when she was 18. Her menses before marriage were of the twenty-eight day type, lasting three to four days, with dysmenorrhœa.

The obstetrical history was as follows :—

First child, not quite full time, was dead-born : instruments were used. There had been no previous history of miscarriages.

Second child, still-born at full time.

Third child, male, full time, lived 11 days.

Fourth child, female, full time, lived $1\frac{1}{2}$ hours.

Fifth child, female, full time, lived $2\frac{1}{2}$ years.

Sixth child, female, full time, alive and healthy, aged 8 years.

Seventh child, female, full time, died of convulsions when 2 months old.

Eighth child, female, full time, alive and healthy, aged 2 years.

Ninth child, present case.

None of the other children were deformed.

The history of this last pregnancy was that she had not menstruated since May, no date of quickening: the birth was not expected until the end of January 1911.

During her pregnancy she had fallen on the floor several times by accident, but did not take much notice of these falls, as she never hurt herself severely.

She affirmed that she was not addicted to the taking of stimulants.

The nurse who conducted the case and reported the birth thought it was a male child.

The delivery was a normal L.O.A.; there was nothing specially noted with regard to the placenta, membranes, or liquor amnii.

The child was weakly, and only survived birth three days.

During life it took the bottle fairly well, and cried, though feebly, at times. When it cried or strained in any way the extroverted area became prominent: the proboscis-like protrusion to be noted later, which was situated in the centre of the extroverted area, straightened out, and normal meconium was extruded.

The urine issued from a small opening placed on the inferior aspect of the area to the right of the middle line.

There was no anal opening, but a slight depression was present.

The extroverted area was somewhat triangular in shape, the apex of the triangle being below the attachment of the cord.

The centre of the area was prominent and cone-shaped, the apex of the cone terminating as a curved proboscis-like structure, the curvature being to the right; at the end of this was an opening through which meconium passed, as already noted.

The upper part of the area was roughened slightly, and blended with the skin of the abdominal wall.

On the inferior surface there was an evagination surface

depression, at the side of which was a slit-like orifice, which was found later to communicate with the large intestine.

The opening through which urine escaped was situated to the right, and on a posterior plane to this eminence.

The inferior surface of the area was rougher than the superior, and presented a more rugose appearance.

There were a few other prominences on the inferior and lateral surfaces, as well as a few openings which were only explained on later dissection, the making of sections, and the use of the microscope.

A post-mortem examination was made of the body, which revealed several interesting developmental anomalies.

A vertical incision from the ensiform cartilage to the umbilicus, and a transverse incision at the latter point, exposed the abdominal cavity without disturbing the extroverted area and its connections.

The stomach and liver were seen to be normally situated. The intestinal coils seen were all of small intestine. There was a small tag of connective and areolar tissue which was the only remains of the great omentum. It was attached to the left side of the greater curvature of the stomach, and lies on a coil of small intestine.

The intestine measured 30 inches from the pylorus to its termination, which was the outgrowth already noted in the centre of the extroverted area. It was attached by a thin mesentery to the posterior abdominal wall; this attachment passed down in the mid line into the true pelvis.

There was no trace of large intestine at first; but on removing the coils of small intestine, a small oval body, yellowish in colour, with a recurved blind extremity was noticed lying in the centre of the pelvic cavity; this proved to be a portion of large intestine.

It was bound to the pelvic floor by fibrous tissue, and its anterior portion crossed the junction of the sides of a

double vagina, to become attached to the extroverted area at the evaginated portion mentioned previously.

Sections made and stained showed that it contained a gelatinous material which stained homogeneously. The walls were typical of large intestine, with the mucous membrane showing tubular glands lined with columnar epithelium, many of the cells containing fat-globules. This part of the intestine was supplied by the inferior mesenteric artery.

The Genito-Urinary Tract.

Fig. 1 shows the relations of the organs after removing the intestines and the peritoneum.

Anteriorly lies a double vagina, the walls of which are joined in the lower half. The portion of large intestine crosses the junction. The right vagina was the larger, and is seen connected with a partially developed uterus with normal broad ligament, Fallopian tube, and ovary. The left vagina had a very rudimentary uterus attached, though the broad ligament, tube, and ovary seemed normal. The right vagina communicated with the surface on the extroverted area by a small opening. On injecting fluid through this opening the right vagina dilated, but the left vagina was unaffected, showing that there was no direct communication between them.

This was confirmed when later a piece of the wall of the left vagina was cut out and the cavity opened up ; no communication could be found even after injecting fluid from this side, nor did the left vagina communicate with the surface. As will be noted later, the left ureter was attached to the posterior surface of the left vagina.

Behind the right reproductive organs, lying at the pelvic brim, is the right kidney. The lower half was in the true pelvis, the upper half projects into the abdominal cavity above the brim. It is flattened antero-posteriorly, so as to be somewhat quadrilateral in shape, and shows lobulation. The right

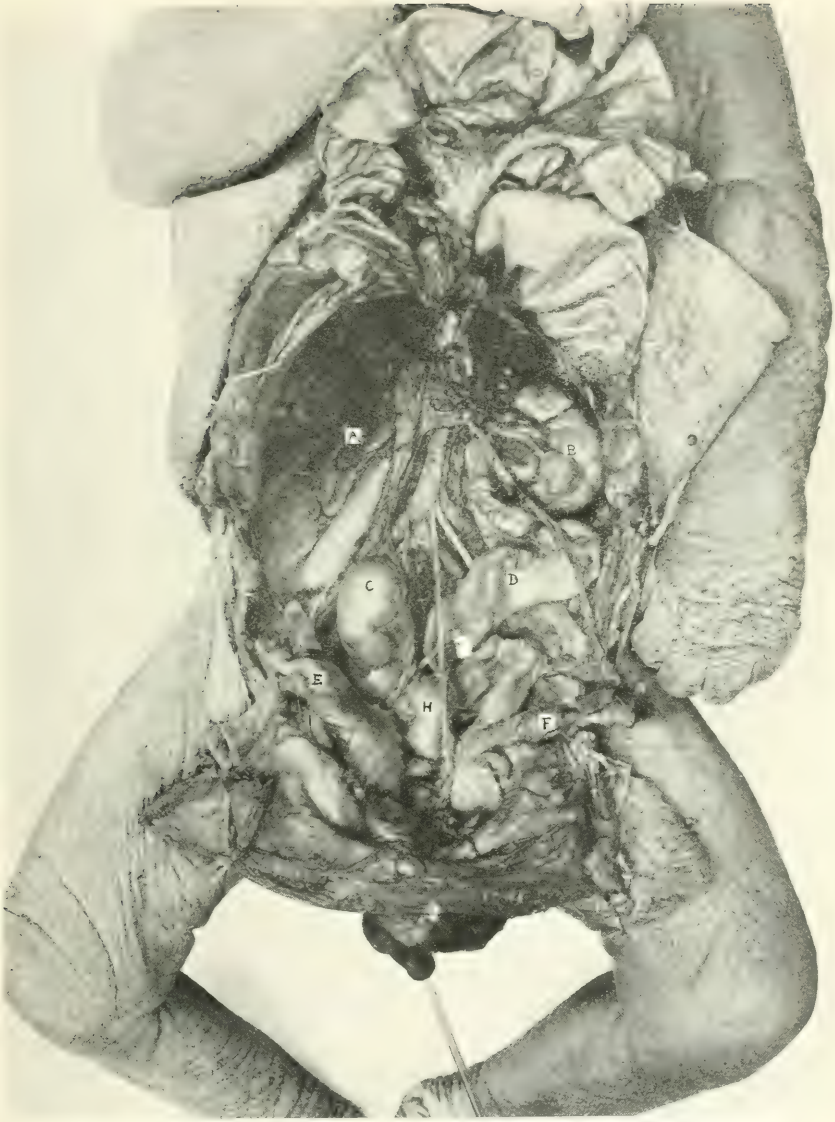

FIG. 1.—Exomphalos in fœtus. Cloacal Vesical Exstrophy in female.

After removal of intestines and peritoneum, showing double vagina, rudimentary large intestine, and kidneys with their ureters.

A, Right Supravenal Gland; B, Left Kidney; C, Right Kidney; D, Left Ureter; E, Right Broad Ligament; F, Left Broad Ligament; H, remains of Large Intestine.

[*Face page* 20.

ureter passed downwards over the lower extremity of the kidney, then turned forwards and upwards laterally to the right vagina, and opened on the surface between two small tubercles.

The arterial supply of the right kidney came from three branches, one from the lower end of the abdominal aorta, the other two from the common iliac on the right side. The venous return entered the lower part of the inferior vena cava.

The right suprarenal gland is seen occupying the normal position of the right kidney. It extended from the diaphragm to the iliac crest, and measured $1\frac{3}{4} \times \frac{3}{4}$ inches.

The left kidney is seen in its normal position. It was smaller than the right kidney, and showed marked lobulation and some cysts on its surface.

The left ureter measured about $4\frac{1}{2}$ inches in length. It was irregularly dilated, the narrowest part being $\frac{1}{8}$ inch in width, the widest being $\frac{5}{8}$ inch.

The lower end was attached to the left vagina on its posterior surface, but on injecting fluid into the ureter no communication with the vagina was found.

The arterial and venous supply of the left kindney were normal.

As it lay, the left ureter looked like an enlarged colon, and was at first mistaken for that.

The left suprarenal gland was more or less in its normal position: it measured $\frac{1}{2} \times \frac{1}{4}$ inch.

With regard to the bones, the symphysis pubis was absent, the iliac and portion of pubic bones being widely separate anteriorly. The vertebræ are all in normal position, there being no sign of a spina bifida.

Sections of different parts of the extroverted area were made and examined microscopically. These showed that while the greater portion of the area was covered by normal bladder epithelium (squamous epithelium), there was an area in the

centre, between the prolapsed end of small intestine and the evaginated part inferior to it, which was covered by normal mucous membrane of small intestine.

The apex of the evaginated part was covered in its lower part by typical mucous membrane of large intestine.

This is a brief description of the case, the chief features of which are—the size of the extroverted area, with the anomalies caused by the extroversion; the condition of the lower end of the intestinal canal; the arrest in development of the reproductive organs; the pelvic position of the right kidney, also the peculiar attachment of the left ureter, which has evidently caused dilation of the ureter and secondary hydronephrosis.

With regard to the origin of this arrest of development of the anterior abdominal wall there are one or two points to be noted; one is the absence of attachment of fœtal membranes to the area, and another is the absence of spina bifida—these are two of the commonest causes given for this condition.

Dr T. B. Johnston, of the University Anatomical Department, has worked up the histology of the specimen, and is about to publish his results in the *Journal of Anatomy and Physiology*. He intends making some suggestions as to the probable cause of the condition.

I wish to express my indebtedness to Sir Halliday Croom for granting permission to publish this case, to Professor Alexis Thomson for permitting me to work in the Surgical Laboratory, and giving me every assistance, to Dr J. W. Ballantyne for his kind suggestions, and also to Mr Frank Pettigrew for the excellent photographs which he took for me.

The President said they were very much indebted to Dr Morison for his paper, and to Dr Johnston for the excellent way in which it was recorded. Anatomical-pathological papers did not lend themselves to much discussion, nevertheless they would be glad to hear any remarks.

Dr Berry Hart said they had had a very excellent clinical record from Dr Johnston of this most interesting deformity. The scientific description was given in the *Journal of Anatomy and Physiology*, where the causation was taken up and the whole matter made very clear. The clinical history, however, had no bearing on the subject, and he thought even the most ingenious anatomist would not consider the mother having fallen on her face as having anything to do with the extroversion.

The difficulties were, firstly, in the lesion itself; and, secondly, the most extraordinary errors into which observers had fallen. Professor Wood thought it was due to an adhesion of the placenta and membranes; but no one had confirmed this. Dr Gemmel and Prof. Paterson of Liverpool had recorded a case where there was no extroversion of the bladder, but where only skin was present below the umbilicus in the middle line. The explanation which Prof. Paterson—the Professor of Anatomy at Liverpool—gave was most extraordinary, namely, that in this case the child had been adherent to the placenta, or rather the placenta to the child, at this area, and that the doctor or midwife had ligurated it off. Then, again, the allantois was blamed as the cause—another extraordinary view. The human embryo has no allantois, all that it has is a small tube in the early umbilical cord, and consequently it can play no part whatever in this deformity.

Dr Johnston had given what he (Dr Berry Hart) thought was the real explanation, namely, that the extroversion is due to the yielding of the cloacal membrane, which is the membrane in early fœtal life lying in front of the entodermal cloaca and in the middle line. The primitive gut is formed by an infolding of the blastoderm, and the bladder and rectum are formed by coronal folds. In the middle line there is thus a weak part where the mesoderm is wanting from the first, or fails to grow in, and it is the yielding of this mesodermic portion that causes the extroversion of the bladder. The

difficult point, of course, was to explain exactly the openings into the small intestine. As Dr Johnston suggested, it was quite possible that the part of the gut in front of which the extroversion arises plays some part in the formation of the small intestine. The ureter and vagina open on the right side, and he suggested that Dr Morison and Dr Johnston should compare their case with Dr Champneys' specimen on this point. But this was rather an abstruse subject, and perhaps he had gone into too much detail. The Society was to be congratulated on having had this able paper read before it.

The President, before calling on Dr Johnston to reply again, thanked him for his and Dr Morison's communication, and assured him it would be a valuable contribution to their *Transactions;* he also thanked Dr Berry Hart for his most lucid explanation.

Dr Johnston, in replying, thanked the Society for the kind way in which his paper had been received. What seemed to him to have been the chief difficulty in the previous cases was that men had been drawn away by the opinion that the extroversion of the bladder necessitates rupture of the ovisac, and they had worked from that standpoint. He assured Dr Berry Hart that he had cut what he took to be the middle portion of the retroverted area, and found it covered with epithelium; under that epithelium there was apparently a piece of vagina, which he believed to be the left vagina, making its way to the surface; whether it got to the surface he did not know, it seemed to fade away.

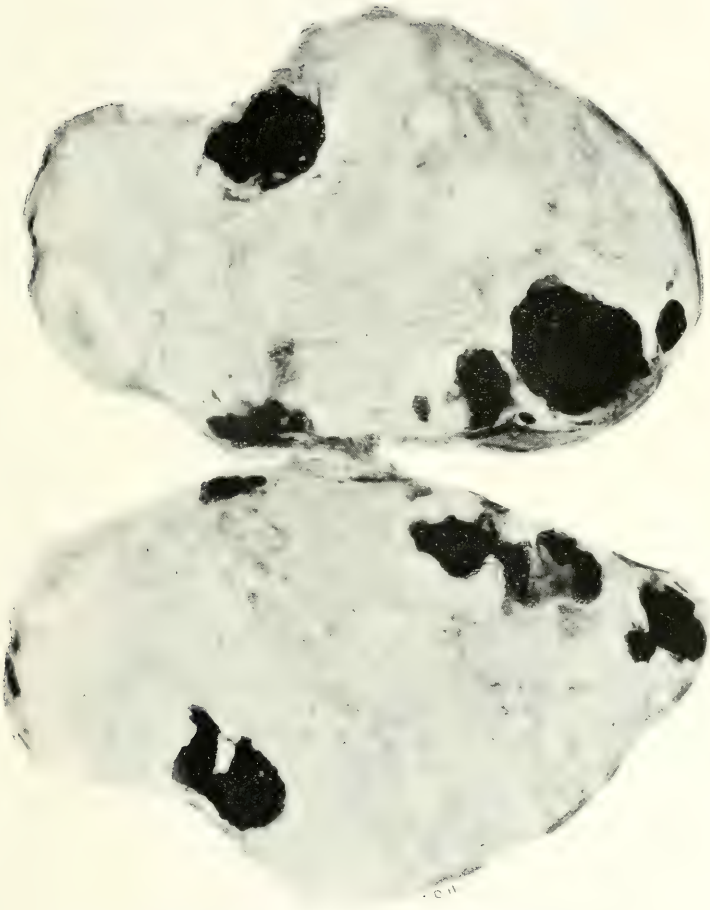

Fig. 1.—Adenomyoma of Uterus, with blood spaces due to menstruation in the endometrial regions.

[*Between pp.* 24-25.

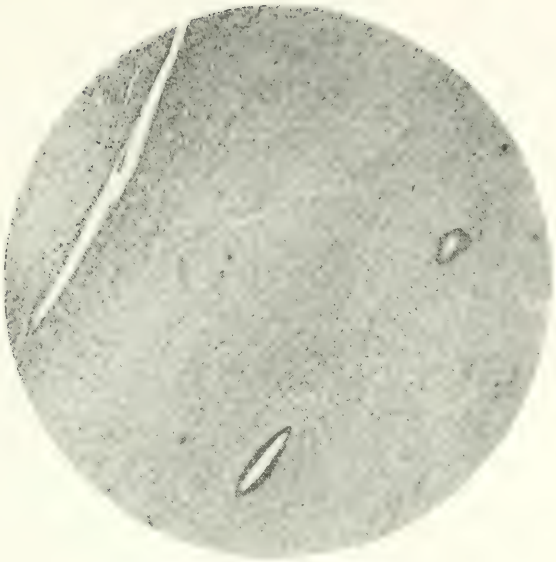

Fig. 2.—Showing gland spaces.

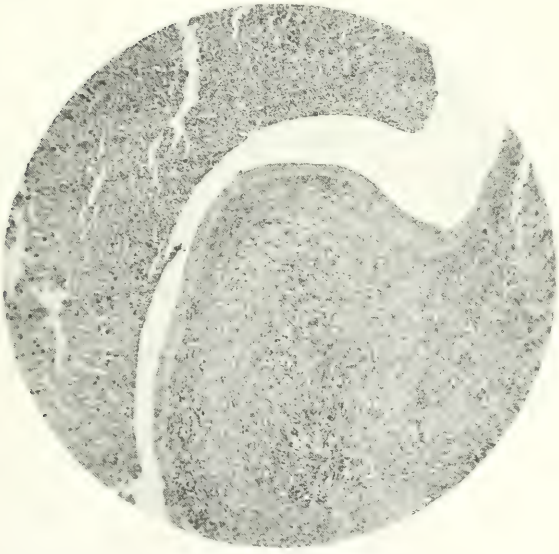

Fig. 3.—Blood-space lined by epithelium, under which is stroma similar to that seen in endometrium.

MEETING II.—DECEMBER 10, 1912.

Sir HALLIDAY CROOM, *President, in the Chair.*

I. *Dr James Young* showed for *Mr Brewis*—FIBROID OF UTERUS WITH HÆMORRHAGIC SPACES (with lantern demonstration). The specimen presented the appearances which are associated with this condition. Here and there scattered throughout the tumour were spaces filled with chocolate-like blood, which obviously had lain in the spaces for some considerable time. It was an adenomyoma with endometrial regions scattered throughout the tumour. These menstruated, and the menstrual blood having no exit, accumulated in the spaces. (Figs. 1, 2, 3.)

Dr Berry Hart inquired about the epithelium.

Dr Young replied that it was quite normal.

Dr Berry Hart asked if it had been recorded before.

Dr Young replied that he had not looked up the literature.

II. *Dr Haig Ferguson* showed—(*a*) UTERINE FIBROMYOMA ASSOCIATED WITH ADENO-CARCINOMA. Miss M'G (æt. 44)—swelling in abdomen for seven years; never noticed any appreciable difference in size latterly, but her doctor saw some increase during the last six months. Had a white discharge for the last seven months, but never at any time tinged with blood. No unpleasant odour. The periods were very profuse. Operation, 15th November 1913. Pathological report—*fibroid* with *adeno-carcinoma.* Kelly and Cullen found adeno-carcinoma as a complication in 1·7 per cent. in a series of 1400 cases of myomatous uteri, the myomatous condition in nearly all cases partly or completely obscuring the presence of the carcinoma. Importance of examining the fibroid after removal, in cases where supravaginal hysterectomy has been done.

In this case supravaginal hysterectomy was done on the

15th November last, and on recognising the condition of the uterine cavity, Dr Ferguson at once proceeded to remove the cervix. The patient's health and appearance have improved extraordinarily since the operation.

(*b*) Fœtus with meningocele, kindly given by Dr John M'Laren, in whose practice the birth occurred. The mother was a primipara, aged 39. There was oligo-hydramnios. No adhesions of membranes to vertex. Apparent complete amputation of one finger. Two fingers tightly strangulated by fœto-amniotic band, showing how their further growth is thereby arrested.

(*c*) Photograph shown from another case — a young woman—showing rudimentary fingers and finger-nails attached directly to atrophied forearm. Skiagram of this case shows absence of carpus, metacarpus, and phalanges, and great shortening of radius and ulna. (Figs. 1 and 2.)

(*d*) Dermoid cyst which had undergone axial rotation. The main portion of the ovary seems to be unaffected, and can be seen quite distinct from the dermoid mass. The Fallopian tube is very œdematous and is the seat of a hæmatosalpinx, the result of the torsion.

III. *Dr W. Fordyce* showed—(*a*) ovary removed eighteen months after previous partial resection. The specimen was interesting in connection with the question of leaving pieces of ovarian tissue. One was very reluctant to remove diseased appendages in comparatively young people. The patient, aged 39, was operated on eighteen months previously on account of a swelling the size of two fists on the right side, while the left ovary was also distinctly cystic. Practically the whole was excised, only a very small portion being left. The patient returned again eighteen months later with distinct swelling on the left side. It was a common experience in the Infirmary in removing diseased ovaries and trying to leave a

Fig. 1.

Fig. 2.

[*Face page* 26.

piece, that the patient's symptoms returned and the other ovary was found to be diseased.

(*b*) TUBO-OVARIAN CYST. The patient complained of sub-acute symptoms and pain, and had a distinct soft cystic swelling. In Bland Sutton's book the condition was described as a hydrocele of the ovary. The fluid could be forced from the ovary into the tube. How the condition arose exactly he always found it exceedingly difficult to explain.

Dr Berry Hart said it was not an ovarian hydrocele. The hydrocele was a pouch of the peritoneum round the ovary.

IV. *Dr R. W. Johnstone* showed—FŒTUS COMPRESSUS IN THE MEMBRANES OF ITS TWIN. He was able to say very little about the specimen, having found it in the old part of the University when looking over specimens. Sections showed that what at first he thought was a tumour was really a fœtus lying in the membranes. A drawing was exhibited showing the two placentas, with the fœtus developed about three months.

V. *Mr Lewis Beesly* showed — INFANT WITH ABNORMAL CONDITION OF LOWER LIMBS, probably of intra-uterine origin. The child, aged one month, was a face presentation, and the birth was in no way unusual. After birth it was discovered that the lower limbs were in an abnormal attitude. The normal attitude of the lower limbs in the embryo is the thighs externally rotated and adducted. In this case the child appeared to have been lying in the uterus with the thighs internally rotated and abducted, with the result that at birth it was quite possible to put the leg backwards into the child's mouth. The child was now improving The legs had merely been tied together and kept extended. When Dr Beesly saw the child two days after birth, any attempt to produce the present condition caused lordosis of the spine. The specimen was brought before the Society in order that he might ascertain from the members if this was an ordinary occurrence.

The President said the case seemed to be quite unique as far as he remembered.

Dr Haultain remarked that an interesting point was the face presentation, which showed a certain amount of extension.

VI. THE PLACE OF GYNÆCOLOGY IN THE MEDICAL CURRICULUM AND IN GENERAL PRACTICE.

By A. H. Freeland Barbour, M.D., LL.D., Lecturer on Gynæcology in the University of Edinburgh.

Among the many important changes brought about by the recent rearrangement of the clinical teaching in the Edinburgh Royal Infirmary, a noteworthy one is the recognition of Clinical Gynæcology as a department by itself. This is the latest step in what might be described as the recent development of gynæcological teaching, and it is the natural result of what had occurred previously. The first step was the recognition of gynæcology as a distinct subject in the systematic course of lectures, distinct I mean from midwifery, which occurred in March 1906, when the University Court appointed a Lecturer on Systematic and Clinical Gynæcology. The next step was taken by the introduction in May 1907 of an examination in clinical gynæcology, this subject thus coming to stand alongside of clinical surgery and of clinical medicine in the final examination. This did not imply that it was of the same importance as the other two subjects for the University degree in Medicine, but it emphasised the fact that no graduate was to be allowed to enter on the practice of medicine without some evidence that he possessed at least an elementary knowledge of this subject. The third and last step has now been taken by the recognition of gynæcology as a special department in clinical teaching, with a Board of its own to direct that teaching. How far this change will modify the arrangements for clinical teaching in the Infirm-

ary which have taken shape during the past seven years remains to be seen. Improvements can only be introduced slowly. The curriculum is now so complex that many other interests have to be considered in conserving and developing one.

It may not be out of place here to refer to the former conditions under which gynæcology was made part of obstetrics in the systematic course in the University, and of clinical medicine in the Royal Infirmary, in order that, while taking advantage of the new conditions, we may not lose hold of what was good in the old.

In the University class-room every fifth lecture of the course of obstetrics was given up to gynæcology; four days a week there was a lecture on midwifery, and on Friday there was one on gynæcology. The reason for this association of gynæcology with obstetrics was twofold—scientific and practical. I use the term scientific in the old sense, when a distinction was drawn between the "science" and "art" of this subject. An inquiry into the factors in gynæcological diseases brings out the fact that not less than one-half of these can be referred to processes which concern the obstetrician. I made an analysis of 2000 cases in the University gynæcological ward, and found that in nearly 50 per cent. the complaint was referred, directly or indirectly, to conception or childbirth; in 9 per cent. to the onset of the menstrual function at puberty; in 7 per cent. to the patient's entering married life; while 20 per cent. began to suffer later in life, towards the menopause. Such an analysis does not account for all the cases, some pathological conditions, *e.g.* certain tumours, having no definite relation to any one of these four events. Looking at this question from the point of view of practice, experience shows that the patients of the working and middle classes, who form the large proportion of the clientele of the general practitioner, expect to be treated in the first instance by the physician who attended them at their confinement. The examination of the patient is in the first

instance made by him; the diagnosis of her condition depends on him; and the routine treatment of chronic cases belongs to him. It usually rests with him to say when the case should pass out of his hands into those of the specialist.

In the Infirmary every sixth lecture of the course of clinical medicine was devoted to gynæcology. The proportion of lectures was partly determined by the association of the Professor of Obstetrics and Gynæcology with the other lecturers in the class of clinical medicine. But if we look at the question from the point of view of the subject rather than the lecturer, we find that there was much wisdom in this association and in the proportion. Dividing the morbid conditions according to the various systems—the nervous, the respiratory, and so on—it may be said that the affections of the reproductive system furnish nearly that proportion of the work of the general practitioner as far as his female medical patients are concerned. Further, however much may be said to-day against this association of gynæcology with clinical medicine, it gave expression to the fact that affections of the reproductive system are so intimately related to affections of the other systems that in the training of the student this relationship must be emphasised. I need not elaborate this point, but would only refer to the close connection of disturbances of the nervous system and digestive system with morbid conditions of the pelvis, and also to the discovery of the part played by the ovarian internal secretion in metabolism. In the recognition of gynæcology as a subject by itself, its intimate relationship with medicine must not be lost sight of. This has been a feature of gynæcology in Edinburgh, not only in the medical curriculum but in practice afterwards. In former days the specialist did not at once enter on this specialty, but took up general medicine as well, and gradually separated himself from that. Specialism was a peak in a mountain range, not a volcanic outburst in the plain. It was the broad base from which they rose that enabled the great

men of last generation to stand out like mountain tops. A wide knowledge of the relation of their own specialty to other departments of medicine, a broad view of the features of the individual case for the time under their care, gave them a sound judgment, which made their opinion weighty.

Passing now from the position of gynæcology in the medical curriculum to its place in general practice, we would ask what is meant by the term "specialty" when applied to gynæcology. Gynæcology is not a specialty in the same sense as mental diseases, or even diseases of the eye. When a case of mental disease presents itself, the practitioner at once recognises that here is a case which does not belong to him. And the same is largely true of diseases of the eye. Putting on one side major gynæcological operations, we might say that specialism in gynæcology is more akin to specialism in diseases of the heart or lungs, in which, by devoting special attention to affections of one system and gaining a wider experience in it than falls to the general practitioner, the physician has come to be recognised as an authority on that system.

Nor should gynæcology, if properly taught, any longer foster in the lay mind the erroneous idea that a special knowledge is required to deal with diseases in this part of the body, a special knowledge as distinct from the experience which comes to the practitioner in the course of his work. The difficulties which beset the clinical investigation of gynæcological cases have in the past sent the graduate into practice inadequately prepared to deal as efficiently with diseases of the reproductive system as with those of the other systems in the female. To this insufficient equipment is largely due the above-mentioned erroneous idea in the lay mind. To it also is due the tendency to what I might call quackery in the treatment of diseases of women, from which no branch of medicine is exempt. A quack, according to Webster, is "an ignorant practitioner"; "one who pretends to skill or knowledge not possessed." The tendency to this in

gynæcological work is partly due to the fact that affections of the reproductive organs cannot be discussed with the same freedom or explained to the patient as clearly as in the case of other organs. And when no lesion is present, it requires a thorough knowledge of his work on the part of the practitioner to tell his patient with confidence that there is nothing the matter, which is the only treatment. Further, in dealing with pathological conditions of other systems, the "ignorant practitioner" can become less ignorant. He can by a repeated examination of the heart or lungs "teach himself" in practice. But my experience is that unless the graduate has been taught as a student in the hospital or dispensary under skilled guidance how to make a bimanual examination, and also what may be learned from it, the little gynæcology he has picked up as a student becomes less, and this may end in "a pretence to skill or knowledge not possessed."

To avert this, gynæcology should not be relegated to a post-graduate course, but form an integral part of the student's hospital training. Hence there has been given to every student during recent years in Edinburgh an opportunity such as he had not formerly of learning the elements of gynæcological investigation, and a clinical examination has been introduced into the final to ensure that he has availed himself of this opportunity. The time at present allotted to gynæcology is inadequate as far as clinical study is concerned, but it is hoped that, in view of the importance of the subject to the practitioner, the present arrangements may be improved.

While gynæcological diagnosis and that part of operative treatment which we call minor gynæcology will continue to belong to the general practitioner, operative gynæcology will become more and more the work of a specialist. It is on its surgical side that gynæcology has of recent years been specialised. The operating gynæcologist must be prepared to deal with any abdominal condition that may crop up unexpectedly in, or form

a necessary part of, an operation on the female pelvic organs. This part of the gynæcologist's work has of recent years become so specialised that the surgeon has claimed the gynæcologist as a member of his craft.

On the other hand, it is interesting to note that while specialisation has developed in what I might call the surgical aspect of gynæcology, in its other aspects there has been a tendency in the opposite direction. In its pathology, for example, the idea used to be current that there was a special pathology of the female pelvic organs, which found expression in gynæcological literature—for example, in such books as Bennet's *Inflammation of the Uterus* and Grailly Hewitt's *Mechanical System of Uterine Pathology*. To-day the general principles of pathology are applied to pelvic disease. It is recognised that, apart from the peculiarities of the anatomical structures involved and their physiological changes, there is no difference between salpingitis and appendicitis, and that epithelioma of the cervix does not differ from epithelioma of the lip. The same idea had crept into clinical work, and came out in the term "diseases peculiar to women," or, as I heard a medical man phrase it, "the peculiar diseases of women." To-day, while on account of the delicate and often difficult nature of gynæcological investigation the gynæcologist retains his place as a consulting specialist for reasons which do not apply to the consulting physician, a considerable part of gynæcological practice is being relegated to the general practitioner.

It is significant that Dr Howard A. Kelly of Baltimore, some eleven years after writing his great work on *Operative Gynæcology*, brought out one on what he calls *Medical Gynæcology*. In the preface, after referring to the development of gynæcology into a major surgical specialty, he says: "This period of surgical evolution is now at last clearly at an end, and I deem it a fitting time to review once more, from our new and advanced

standpoint, the relationship of our specialty to the field of general practice.

"To my mind, the evolution of scientific medicine must ever run this course: the general practitioner yields up to a little group of investigators that portion of his territory which is most obscure and difficult, in which he has made the least progress; the field is diligently cultivated, and a specialty is formed. Then in time the specialist so simplifies the etiology, the diagnosis, and the treatment, that he is able to hand back a part at least to the general practitioner. . . . It will be my effort in the following pages to review my special field, in an endeavour to return to the general practitioner that portion of it which he ought to recover by right of his prior lien."

This is a significant statement as coming from one who has done much to develop gynæcology on its surgical side. To it we might add that if the period of surgical evolution is at an end we seem to be at the beginning of a period of medical evolution. The discovery of the internal secretion of the ovary is making the operator hold his hand in the removal of that organ; the value of organic extracts, thyroid, suprarenal, and pituitary, is becoming recognised, and they are being used in gynæcological cases; radium is taking its place alongside of the knife in the treatment of cancer.

In delimiting the surgical area in gynæcology these two considerations should be borne in mind. When operative gynæcology was in the hands of the surgeon, as at one time in Paris and also in London, he called in the gynæcologist to make a diagnosis. Experience shows that too great freedom with the exploratory incision has a tendency to weaken other methods of diagnosis. Lawson Tait's *dictum*, "Open the abdomen and see," while it showed his strength as a surgeon, was a confession of weakness, for with it was associated a lack of appreciation of the value of the bimanual method of examination. Again, while not undervaluing the significance of surgical methods in

the relief of suffering, we must be on our guard against too short cuts to health, which have not always proved satisfactory in the long run. The fact that a thing may be done with impunity is not a sufficient reason for doing it.

To return to the point from which we started, we might thus describe the present situation. The independence which gynæcology has claimed of late, and which has found expression in the changes in the curriculum, is not the independence of standing alone. She still holds out one hand to the specialist and the other to the general practitioner. She has her grand manner, in which, as hospital surgeon, she performs brilliant operations; yet I am not sure but she is as great when, in the guise of the family physician, she cares for the health of the mothers and growing daughters of the community. She is equally at home with the brilliant operator and the sagacious practitioner; and when the honours go round, will the greater fall to the operator in his theatre furnished with every appliance and waiting for his patient to be wheeled in, or to the country practitioner starting on his lonely drive across the moor?

The President said Dr Barbour had given a very masterly paper on the whole subject.

Dr Berry Hart said they had listened to an exceedingly good and very carefully written paper, and with a great deal of what was said he agreed.

With regard to the development of gynæcology, the question was whether gynæcology developed from obstetrics or from medicine. He thought gynæcology evolved from obstetrics, because it was the obstetrician who was in time past consulted first. Dr Barbour put forward an exceedingly interesting explanation of why the gynæcologist is ranged with the physician in the Edinburgh School. He (Dr Berry Hart) thought the practical explanation was that in Edinburgh the

gynæcologist was always a physician and a Fellow of the College of Physicians; indeed, it was only a Fellow of the College of Physicians who up till recently could be a gynæcologist in the Royal Infirmary. Dr Keiller was a physician and had medical wards at one time in the Royal Infirmary. As he was most interested in diseases of women, he proposed to his colleagues that he should devote himself to them, and accordingly he took charge of a medical ward for this purpose. That, he believed, was the practical explanation of how gynæcology became the province of the physician for a time. The late Sir J. Y. Simpson had also a ward for diseases of women, but how this originated he was unable to say. At anyrate, Sir James was a Fellow that was one time President of the Physicians.

As to whether the gynæcologist should be a surgeon or a physician, Dr Barbour had stated the matter very judiciously. No doubt for a long time gynæcology became a surgical specialty; now they had electrical and other methods which, to a certain extent, might supplant that, but he thought that for a long time a great proportion of the treatment in gynæcological cases would be of a surgical nature.

There was only one point which he thought Dr Barbour handled delicately but a little unjustly, namely, that gynæcology lent itself to quackery. Dr Berry Hart did not think so. *Ignorance* lends itself to quackery; and if a heart or chest or brain man is ignorant, he will become a quack just as easily as an ignorant gynæcologist.

In conclusion, he agreed in thinking that gynæcology had no special pathology, it was just the pathology of the other parts of the body.

A curious point in the history of the pathology of diseases of women was that at one time it was really of a philosophical cast. Spinoza attempted by the definition of substance and its relations to elucidate the world and the relations of God

and man. In the same way, Henry Bennet, Emmett, Hewitt, and even Lawson Tait tried to make gynæcological disease arise from one disease—the cervix, the ovary, or the tube. This lasted until operative gynæcology gave specimens for accurate investigation, but the idea still lingered among us.

Dr Barbour's paper was a sound one, and he was to be congratulated on attempting to solve a most difficult problem, clinical instruction in gynæcology.

Professor Harvey Littlejohn had come to learn, and did not think he had anything to say, because Dr Barbour had not trenched on any debatable ground. He thought all admitted that there was debatable ground in regard to the position of gynæcology in the course. There was, he supposed, no question that it was a very important branch indeed of medicine for the general practitioner, and that, if possible, every graduate who goes out from Edinburgh should have a good working knowledge, at least sufficient to prevent him from doing any harm. For that purpose he had gathered from Dr Barbour that the teaching at present was almost insufficient, and that he would like to see a longer course for the ordinary student in the University. Personally, his feeling with regard to all specialties was that they should get as much time as possibly could be given. They could not turn out men too well trained. Unfortunately, however, there were only five years, and therefore they had "to cut the coat according to the cloth." He had come to the meeting because this question of whether more time could be given to specialties —if one could call gynæcology a specialty—and whether the attendances of students could be systematised at these classes so that they got the maximum of knowledge in the minimum of time, was under discussion. The Faculty at the present time was considering whether they could not economise in certain directions and give more time to others, so as to

improve the training of the student at the end of five years. Dr Barbour, Dr Brewis, and he were agreed on the scheme which would be laid before the Faculty. His object in being present was to learn; he had learned a great deal, and would learn still more if those present would give their views.

Dr F. W. N. Haultain had listened with great interest to what Dr Barbour said. Personally he felt that there had been far too much tendency to separate gynæcology from obstetrics. In the first place, why separate two subjects so interwoven? As Dr Barbour had said, probably 50 per cent. of gynæcological cases owed their origin to obstetrics. He thought to separate the theoretical teaching of these two subjects, given by different men, was undesirable. The continuity of the teaching of obstetrics into that of gynæcology was to his mind perhaps best. To take an example, suppose Dr Barbour and he (Dr Haultain) were united in teaching the two subjects; one of them might say to the students that early rising in the puerperium was an advantage, while the other said that to get up early meant sudden death. What was the student to believe? Continuity was, he believed, better. Further, it was unnecessary to have two men, for there could be no doubt that the obstetric physician must be a gynæcologist in order to live; he teaches obstetrics, but he practises gynæcology. Obstetrics would not keep any man; if he had enough cases to keep him he would not have a life worth living; the obstetrician is a gynæcologist, and by gynæcology he keeps a roof over his head. Now, such being the case, when one comes to the question of the gynæcological part of it, he rather fancied under present conditions the student was either getting too much or too little; that is to say, he was being hall-marked when he passed at the University as having passed an examination in gynæcology. Gynæcology he (Dr Haultain) regarded as a specialty as much as diseases of the eye or ear, and not only

so, it was a specialty that was much more difficult to teach because of want of sufficient material; and if one could not give the students sufficient material to make them thoroughly expert in gynæcology, he thought they had no right to turn them on to the public hall-marked as gynæcologists. His impression was that the old-fashioned style, when a certain amount of gynæcology formed a part of the clinical medicine teaching, was preferable.

Dr Haig Ferguson had listened with much pleasure to Dr Barbour's paper, and had little to add to what had already been said in the discussion.

In considering the question of gynæcology in its modern aspects, it can be looked upon from three points of view:—

(1) The abdominal aspect, which is entirely surgical gynæcology, and which is practically entirely in the hands of the specialist;

(2) Vaginal gynæcology, which is partly in the hands of the specialist and partly in those of the general practitioner; it is also chiefly surgical in its aspect; and

(3) Medical gynæcology, which is in the hands of the specialist as well as in the hands of the general practitioner.

The question has recently been raised—Are we gynæcologists justifying our existence, and are we entitled to be regarded as a separate entity? The answer to this is simple, and has been dealt with satisfactorily by the late Dr Arthur Wallace, who said, "So long as there is forthcoming, work which the gynæcologist can accomplish better than anyone else, so long will he survive."

There is no question as to this in vaginal and medical gynæcology, but there is some room for argument as regards abdominal work. The general abdominal surgeon has begun to

think and to act "imperially" in the abdomen, and there is a tendency for the proficient gynæcological surgeon to do the same in some quarters.

There was an increasing tendency to encroachment, not only in this country but all over the world, excepting, perhaps, Germany. Pozzi has said that "there are no pure gynæcologists in France. Every surgeon does all he can in gynæcology, and the younger generation of accoucheurs does the same. It is certainly somewhat difficult to establish strictly a dividing line between these three branches of the medical art—surgery, midwifery, and gynæcology. One might, however, define the limits with a little goodwill, good faith, and reciprocal delicacy. These are virtues which are becoming more and more rare among us, unhappily."

In America we are told that the general surgeon does all the gynæcology he can, and the gynæcologist aims at losing his title and proposes to deal with the abdomen, irrespective of sex.

As Dr Barbour and Dr Berry Hart pointed out, the present position of abdominal surgery was greatly due to the operating gynæcologist.

There can be no doubt that the abdominal surgeon who is also trained in gynæcology, or the gynæcological surgeon who is proficient in general abdominal work, are the class of men who are now to evolve. The tendency has been for general abdominal surgeons, while reaping the advantages, to be somewhat forgetful of their obligations to gynæcology.

With regard to the points which Dr Haultain touched upon, viz. the relationship of gynæcology and obstetrics, Dr Ferguson feared that their separation had become almost inevitable, *i.e.* the separation of the operating gynæcologist from the obstetrician, In some ways this was to be deprecated and very much regretted. There was no doubt that a knowledge of obstetrics had been a great help to the gynæcological surgeon, especially in questions of diagnosis, and ignorance of obstetrics is certainly a handicap

to the abdominal surgeon, from many points of view. It would be exceedingly unfortunate for obstetrics if obstetricians did not receive some training in surgical gynæcology, for the surgical instinct is much needed at present in the domain of midwifery.

So far as the changes in the teaching arrangements, which had recently taken place in our University and Infirmary, were concerned, and which Dr Barbour had described, Dr Ferguson believed that they would undoubtedly result in distinct advantage, both to the general practitioner and to the gynæcologist.

Dr W. Fordyce would have preferred a general practitioner to speak. He thought they had departed from the important subject of the paper, which dealt with the position of gynæcology in the medical curriculum. As a teacher and examiner he had been struck with the difference in the knowledge of the man who had done even a very little gynæcology in the wards and that of the man who had got his learning merely from books. In teaching gynæcology, even in the short time at one's disposal, one could do a great deal in helping a man as a general practitioner. One could not expect to make a student a fine diagnostician. If he could form an opinion that there is or is not a definite lesion in the pelvis, that man, he thought, had gone a great way on the road to gynæcology.

Then Dr Barbour also brought out that in general practice later, one could not gain a knowledge of diagnosis in gynæcology as one could in other departments of medicine; therefore he (Dr Fordyce) thought very strongly that gynæcology should be made a very important part of the curriculum. He was sure they could do that without being, as Dr Haultain said, specialists. If a man went out into general practice and did not give an opinion when called to a heart case, but said, "you must go to a heart specialist," and the next case that

came up was one of indigestion, and he said, "you must go to a stomach specialist and have the stomach contents analysed," it would not do; the same with gynæcology. The general practitioner should be able to recognise early cancers, etc. A man could learn a great deal in a short course of five or six weeks if it were made compulsory; up to the present time it had been voluntary—the students were not made to attend, and therefore they did not take advantage of their opportunities.

Dr Keppie Paterson thought that Dr Barbour had estimated the proportion of gynæcological cases met with in the average general practice at too high a figure. This applied to women being attended for purely gynæcological troubles. With regard to the preparation of a student for general practice, one wanted to give him an ordinary knowledge of all the special subjects. One could not turn him out with a special knowledge of every special subject. The teachers could only do their best in the time available, but he should have sufficient knowledge of special subjects as to recognise urgent serious conditions. The aim of the curriculum should be to give the student a primer knowledge in lectures of all the special subjects. With regard to gynæcology, every man should have a course of lectures and be trained carefully and methodically how to make an examination. The great problem was the lack of opportunities to obtain facility of diagnosis. The student could not obtain experience of the diseases, as he could not get the cases. But if he had been trained to examine a patient properly, he could use his own brains afterwards.

Dr Barbour Simpson said gynæcology could not have too strong a place in the curriculum. One would have liked the general practitioners to give their experiences. The advances which had been made in recent years had no doubt been due to more careful study by the students and their teachers. Large abdominal tumours no longer came under observation.

Dr Haig Ferguson remarked that the separation of obstetrics and gynæcology had become inevitable. With this Dr Barbour Simpson could not agree, gynæcology could never be divorced from obstetrics.

Dr Young, after endorsing the remarks of previous speakers as to the interest of the paper, said, in connection with the question of the most efficient way of teaching, with the comparatively small amount of material which was obtainable in Edinburgh, that it had always been very strange to him that the material which they had in Edinburgh in the hospital in the shape of out-patients, was not utilised. One of the first things which struck one on going round the cliniques in London was the fact that even the meagre out-patient material which they have in the various hospitals was made use of in the most practical manner. To instance one hospital, with which he was familiar—St Bartholomew's. Every Saturday morning Dr Griffiths holds an out-patient clinique. The students go up at 9.30, and each one is presented with a sheet which includes all the various items which it is necessary to note; he then interviews the patient allotted to him—there are about twenty men altogether. By the time the investigation is over the Chief has arrived. The students then take their seats in the out-patient department, and one case is brought in after another. The student reads the history, is asked about the possibility of diagnosis, and then the treatment is discussed. The result was that these men had a working knowledge of gynæcology which, he was afraid, very few students of Edinburgh possessed when they obtained their degree. Why a system of a similar nature was not introduced in Edinburgh, he did not know. The excuse that it was more difficult to utilise Scottish patients for such a purpose than London patients, was one which did not bear very serious scrutiny.

Dr Oliphant Nicholson said he was quite sure that the

doctor who had followed general practice for a good many years made a much better gynæcologist than the man who had had no such training. The same statement applied to every specialty—eye, ear and throat, skin, etc. When he commenced general practice, twenty-two years ago, his knowledge of gynæcology was practically nil. He came to Edinburgh for a short time, and under the President's tuition learned to recognise the gynæcological conditions commonly met with in practice. The President, at that time, taught a few graduates and senior students how to make the bimanual examination, and demonstrated the methods which were necessary to enable one to diagnose uterine displacements and diseases of the pelvic organs. He agreed that very often the really important thing for the general practitioner was to possess sufficient knowledge to enable him to know when to send his patient to the specialist. He thought Dr Barbour had done a great deal of good in emphasising the fact that a practical knowledge of general medicine was an important matter for the gynæcologist, unless he devoted himself entirely to operative work. In a case of amenorrhœa the gynæcological surgeon would be competent to diagnose the underlying cause as an imperforate hymen or a fibroid tumour, but it would require a gynæcological physician to discover that the symptom was dependent upon leucocythæmia or a thyroid inadequacy.

Dr Armour thanked Dr Barbour for his paper, the title of which had suggested to him (Dr Armour) a very interesting discussion, and he had not been disappointed. It was a subject which must have been in the minds of a good many general practitioners. Personally, he had always been thankful that he was led to have a little special training in this department, and he owed to the President and Dr Barbour a very considerable amount of the knowledge he had of the subject. After all it really came to this, if one divested one's mind of the

distinction between special and general work, that they wanted their students on leaving the University to have sound practical knowledge of the methods of examination; whether in gynæcological or heart cases or any other, they should be accurately trained on the points of clinical examination. They had to leave it to the men themselves and their opportunities afterwards, as to which branch of work they would follow.

He agreed with Dr Barbour that there was a little tendency for a man to become more or less a quack in diseases of women. One might, of course, have quackery in any department; but if a man knows very little gynæcology, because of the character of the subject he tends gradually to deteriorate clinically. But he may be constrained to express an opinion all the same. He felt very strongly that it was important every student should have a sound knowledge of gynæcological methods of examination.

He did not agree with Dr Keppie Paterson that most of the general practitioner's female patients were not gynæcological cases; on the contrary, almost every case that came to them was to some extent a gynæcological one; that element was almost always involved in the patient's illness, and one ought to know how to deal with it.

The President congratulated Dr Barbour on having introduced a very interesting discussion. Dr Barbour and he were so intimately associated in the University that it would scarcely do for him to express any different opinion; but fortunately he had no desire to do so, because he was in complete accord with all that Dr Barbour had said. Dr Barbour, in his opinion, had given a most fair and just exposition of the whole matter. His description of the evolution of gynæcology was extremely interesting, tracing how it began in medicine, then went completely over to surgery, and the trend again back to medicine. In his, the President's own experience, it was a curious thing

that he first of all joined the College of Surgeons, and then afterwards, in order to be put on the gynæcological staff of the Royal Infirmary, he had to join the College of Physicians. Now again he was entirely connected with the College of Surgeons. Of course, the great and signal advances in gynæcology had been in surgery, and so far as surgery is concerned, gynæcology was nearly a complete art. He did not believe that gynæcology would ever be absorbed into general surgery. It certainly would not be for its advantage if it were so. Personally he believed that gynæcology and obstetrics should remain intimately associated one with the other. They ought to be taught as one entire subject. Dr Haultain said that they were separated now. From that he begged entirely and absolutely to differ. Dr Barbour and he taught the subjects as one consecutive whole. He did not believe that any one man could efficiently do the work of a Chair of obstetrics and gynæcology himself. He had had experience of it when he succeeded to the Chair which his predecessor, Professor Simpson, adorned; and he would take the opportunity of saying this now that his entire day, from early morning till evening, was taken up with the duties of systematic teaching, clinical teaching in the Royal Infirmary, operating, and obstetric clinical teaching in the Maternity Hospital in the afternoon. He was of opinion, putting himself and Dr Barbour out of the question altogether, that the system adopted in the University of Edinburgh was the correct one. It was only possible to teach this subject thoroughly by two men united in the matter, and who taught in close relation to one another. He had been told that one man could conduct the two subjects personally, by relegating part of the teaching to assistants. He would like to ask wherein lay the difference. It was better that the class should be taught by two men of experience than be handed over to younger and less responsible teachers. The two subjects, gynæcology and obstetrics, should be absolutely undivided,

although both purely surgical subjects, and he desired to repeat that he thought the arrangements of gynæcology and obstetrics in the University of Edinburgh was ideal. He did not want to detain the Society any longer, but thought with regard to the teaching of gynæcology, Dr Barbour had given a satisfactory exposition, and drawn a nice distinction between gynæcology as a specialty and as a matter of general practice; because, notwithstanding what Dr Keppie Paterson had said, it did come into the work of general practice to a very large extent. He congratulated the Society on the discussion, and thought the subject had been very thoroughly ventilated.

Dr Barbour thanked the Society for the discussion of his paper. He was sorry that they had not had criticism from the general practitioner in the country, whose patients have not the same opportunities of seeing specialists. Country practitioners had told him how they feel the need of this special subject; and the appreciation of it in the post-graduate teaching showed how a graduate of former years felt the need of it.

He was obliged to Dr Hart for his contribution to the development of gynæcological teaching. He thought Bennet's and Tilt's work was of a different quality from Hewitt's. He thanked the Dean for the trouble he was taking in meeting the wishes of Mr Brewis and himself as to the teaching arrangements. All that was asked was that more time be given to clinical gynæcology, to enable the student to master the elements of gynæcological examination.

He was afraid there was not sufficient out-patient material at the Royal Infirmary to have an out-patient department, as had been proposed by Dr Johnstone and Dr Young. The conditions were different from the London hospitals where this was being done. He hoped that the dispensary teaching would be continued in Edinburgh; and he advised students to make

use of their limited cliniques also. As regards the Infirmary, the out-patient material could best be utilised at the wards.

He agreed with Dr Haultain as to the advisability of keeping gynæcology united with obstetrics in the systematic course. It was interesting to see that where the subjects had been separated they were again being united, as in Toronto and Montreal. On the other hand, he thought it a mistake to have the whole department under one head, as it was impossible for one man to give the same attention to the clinical teaching of gynæcology when he was responsible for obstetrics as well. He thought the present arrangement in Edinburgh the most satisfactory arrangement of a difficult problem.

MEETING III.—JANUARY 14, 1914.

DR BARBOUR SIMPSON, *Senior Vice-President, in the Chair.*

I. The following gentlemen were elected Ordinary Fellows of the Society:—J. N. Jackson Hartley, M.B., Ch.B., The Residency, Royal Infirmary, Edinburgh; D. J. Guthrie, M.B., 8 Strathearn Road, Edinburgh; A. G. M. Grant, M.B., Ch.B., The Bank, Newtown, N. Wales.

II. *Dr R. W. Johnstone* showed—WAX RECONSTRUCTION MODEL OF PORTION OF EARLY HUMAN OVUM. Professor Arthur Robinson had made the drawings and cut out the wax plates, which, unfortunately, instead of being put one above the other had been put one behind the other. The basis represents the mesoderm; at one point vesicle No. 1 with its attachment to the mesoderm is shown, and vesicle No. 2 with its very definite attachment in the other corner. At no point did the vesicles come into contact with each other, and unfortunately both were incomplete.

III. *Dr Haig Ferguson* showed — ADENO-CARCINOMA OF UTERUS. The patient, a multipara, æt. 59, weighed 16 stones. It was unusual to have a cancerous condition in a woman of that weight. The abdominal wall being 9 inches thick, the vaginal operation was performed.

IV. *Dr W. Fordyce* showed—A CASE OF PERFORATION OF THE INTESTINE BY BONE (thought at first to be a gynæcological case). The patient was sent into Leith Hospital with acute abdominal symptoms, supposed to be gynæcological. There was an acute abdominal condition. Dr Lackie also saw the patient, who was acutely ill. The abdomen was opened in the expectation of finding an appendix. The patient was taking

the chloroform very badly. The bowel burst out, and something was seen to be projecting through the bowel at both sides. Subsequently it was found that there was a large piece of bone perforating the intestine on each side. The abdomen was filled with fluid from the perforation in the small intestine, and the case looked very hopeless. A tube was inserted and the patient kept propped up; she did very well. Dr Fordyce understood that before admission the patient had had a large meal of Irish stew.

V. CONTRIBUTION TO THE STUDY OF THE EARLY HUMAN OVUM, BASED UPON THE INVESTIGATION OF (1) A VERY EARLY OVUM EMBEDDED IN THE UTERUS, AND (2) A VERY EARLY OVUM EMBEDDED IN THE INFUNDIBULUM OF THE TUBE.

By R. W. Johnstone, M.A., M.D., F.R.C.S.E., Assistant to the Professor of Midwifery in the University of Edinburgh; Extern Assistant Physician, Royal Maternity Hospital; Obstetric Physician, New Town Dispensary; Gynæcologist, Livingstone Dispensary.

Our knowledge of the development of the human ovum during the first weeks of its life is still so incomplete that it becomes a duty to place on record the main features of any such specimen that may be met with. From the nature of things these specimens must be rare, and the majority of them will probably always continue to be obtained in early abortions. Although the great possible value of such cases is evidenced by the flood of light that the Bryce-Teacher ovum has thrown on the subject, and by such a record as Jung's, still any specimen obtained *in situ* must of necessity approach more nearly to the normal. I consider myself peculiarly fortunate, therefore, in having obtained within ten months two specimens of an early ovum *in situ*. Moreover, the fact that one is embedded in the uterus and the other in the tube affords an interesting contrast

and comparison of the earliest stages of development in the normal and abnormal situations.

The first specimen is that of an ovum of about fifteen days embedded in the uterus. Its general features do not differ in any great degree from the classical description of Hubert Peters. The embryonic structures are, however, very difficult to interpret, and one perfectly legitimate view of them is that the specimen is one of uniovular twins in a very early stage of development. If such be the case, the specimen is absolutely unique.

The second specimen is an infundibular tubal pregnancy of about twenty days' duration. So far as I have been able to ascertain, this specimen is also unique, all the other records of the very early stages of tubal implantation being based upon ampullar or isthmic pregnancies. In addition to this the case has one other feature of great importance etiologically, namely, that it affords a perfect demonstration of the fact that the ovum must have come from the opposite ovary and migrated round the back of the uterus to reach the site of its implantation.

I have thought it well to divide this paper into two parts. In the first place I have recorded the history and minute anatomical features of both specimens. In doing so I have tried to state what I saw, without reading into the statement my own opinion as to the nature of the appearances. The second part of the paper is a discussion of the different features, in which I have ventured to state my own views as to the specimens, and have compared their most interesting features with some of the other early ova on record.

Terminology.

For the sake of brevity and to avoid misconception, it may be as well to state shortly the sense in which I shall employ certain terms. The terms used to denote the topographical and

histological divisions of the decidua, such as *basalis*, *capsularis*, *vera*, *compacta*, and *spongiosa*, require no explanation. The terms *equatorial* and *polar* applied to the decidua capsularis are explained in the text. Some confusion, however, exists in regard to the meaning of such words as *syncytium*, *trophoblast*, *trophoderm*, and the like.

The word *trophoblast* was applied by Hubrecht to the chorionic or extra-embryonic ectoderm. But strictly speaking it applies to it only when that structure remains in passive relationship to the surrounding tissues. Whenever it proliferates and assumes histolytic functions it ceases to be in line with Hubrecht's definition, and for this proliferative histolytic "trophoblast" Minot has suggested the term *trophoderm*. In man, unlike many other mammalia, this layer is always actively proliferative, and accordingly the term *trophoderm* is the more correct one to use in human development. I agree with Herzog that etymologically either term is a misnomer. It suggests that the layer is wholly connected with the nutrition of the embryo. But, as Herzog points out, the trophoderm is thousands of times greater in mass than the embryo, and this seems too extravagant a provision for nutrition alone. Indeed we have clear evidence that nutrition is only a minor function of this layer in the early stages, and its most important action is to enable the ovum to form stable attachments for itself to the maternal tissues. This it does by proliferating freely, and, in all probability, secreting a proteolytic ferment which destroys and digests the maternal tissues with which it comes into contact.

The term *syncytium* has been so long applied to the outer layer of the trophoderm as to be wedded to it. Etymologically it also is a misnomer, as the word connotes a tissue which is formed by the running together of cells. That this is the real mode of its formation we have no clear evidence; indeed there is as much reason to believe that it arises from the very

beginning as a plasmodial layer. The term *plasmoditrophoderm* would be more correct on this latter understanding. Since, however, we have no proof on either side, and since the word *syncytium* is so constantly used in the literature, I have preferred to retain it. As will be explained in the text, the term *symplasma* is used to denote a clearly degenerative coalescence of cells, or a degeneration of the syncytium.

First Specimen.

On 16th March 1913, a married woman, æt. 29, died suddenly in this city. She had previously complained of no symptoms beyond a shortness of breath on exertion, and a tendency to faintness. She had had three children, the youngest a year old. The husband did not know the actual date of the last menstruation, but he was certain that she had not missed a menstrual period, and neither of them had had the slightest idea that she was pregnant.

A post-mortem examination was ordered, and in the course of it the uterus was noticed to be somewhat enlarged. It was accordingly removed for further examination. The pathologist's notes on the case are briefly as follows:—The heart was in a state of advanced fatty degeneration. The stomach was congested and empty. The liver, spleen, kidneys, and lungs were normal. The uterus was slightly enlarged, and soft. The tubes were engorged with blood. The right ovary was swollen and congested. The cause of death was, therefore, the sudden failure of the heart, owing to advanced myocardial degeneration.

Unfortunately the autopsy was not made until nearly forty-eight hours after death, and the uterus was thereafter kept in Kaiserling's fluid for eighteen hours. It was then opened, but still other twelve hours elapsed before it was seen by Professor Harvey Littlejohn. He at once realised the

possible significance of its contents, and sent it over to Dr Barbour at the University Gynæcological Laboratory. In the absence on holiday of Dr Barbour I began the investigation, and he has very kindly allowed me to continue and complete it.

The uterus was slightly enlarged, and the endometrium very distinctly thickened and thrown into irregular elevations and furrows. On the posterior wall of the cavity, a little above the centre, there was a small dark crimson object, protruding like a wart from the surface. It measured 4·5 mm. vertically, and 5 mm. from side to side. Its base was slightly narrowed, so that it overhung a little at its lower edge. In appearance it suggested a pedunculated wart, or a subepithelial hæmatoma, although the surrounding mucosa was not unduly congested (Fig. 1).

A portion of the endometrium on the opposite side of the cavity was removed and embedded in paraffin. On section it showed the characteristic appearances of a decidua. The projection with its immediately surrounding decidua was thereupon removed, right down to the muscle wall of the uterus; and after I had obtained the advice and guidance of Professor Arthur Robinson on the specimen, it was dehydrated and embedded in paraffin. The entire block was then cut into 679 serial sections, each section being 10 microns thick. The plane of section was vertical to the surface of the decidua, and in the long axis of the uterus. The sections were stained with hæmatoxylin and eosin.

The projection was found to contain a young ovum surrounded with early villi, lying in an implantation cavity filled with maternal blood, and bounded on all sides by decidua (Fig. 3).

Examination of the sections revealed the fact that the specimen had unfortunately received considerable damage—probably due to the uterus being firmly grasped antero-posteriorly during its removal from the body. There is

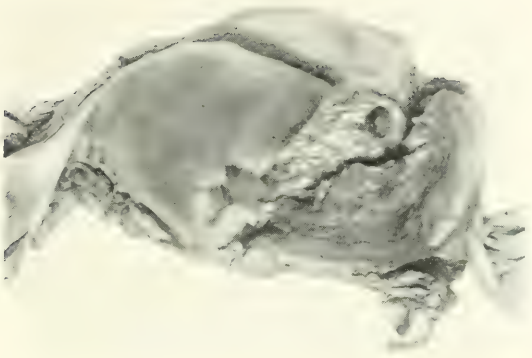

Fig. 1.—Uterus opened, showing ovum *in situ*.

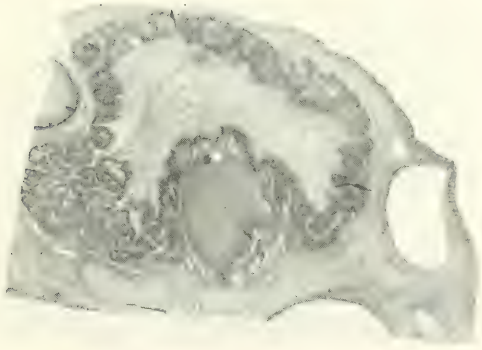

Fig. 2.—Corpus luteum.

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FIG. 3.—Section showing blastocyst and intervillous space.

B—Mesoderm of blastocyst shrunk away from epithelium in many places.
Bl—Blastocyst.
BS—Blood space.
CE—Chorionic epithelium.
DB—Decidua basalis.
DC—Decidua capsularis.
E—Embryonic vesicle with attachment to chorionic mesoderm.
IS—Intervillous space.

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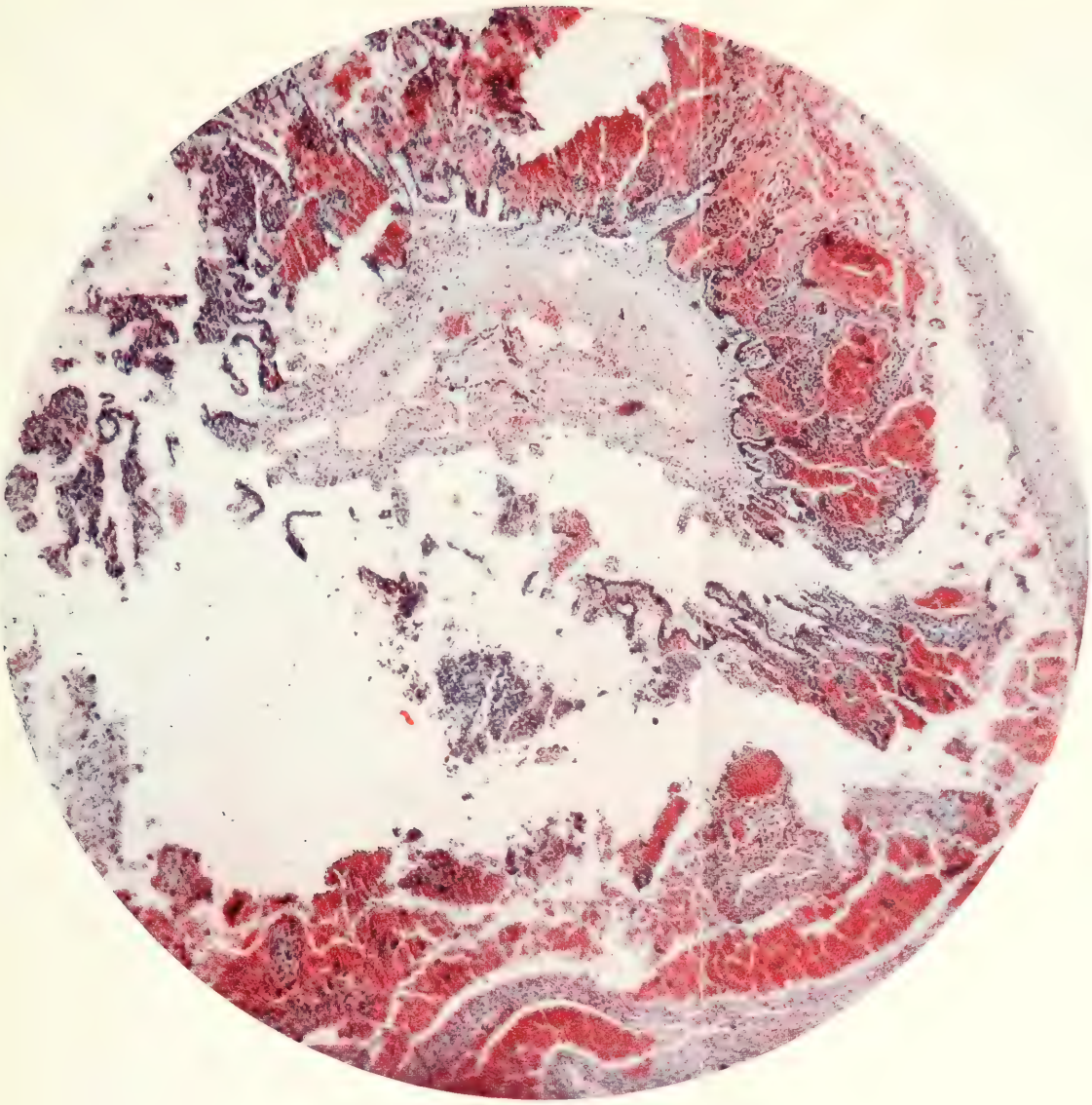

thus a rent in the decidua capsularis, which has also extended into the wall of the blastocyst or chorionic vesicle. The latter contains some maternal blood, showing that the damage had been received before the fixation of the specimen. Apart from this the specimen is in a wonderfully good state of preservation considering the circumstances in which it was obtained. The histological appearances are not to any extent obscured.

Dimensions of the Ovum.

Owing to the fact that it is ruptured, it is impossible to give absolutely exact measurements of the implantation cavity. But as the amount of displacement caused by the rupture is comparatively slight in most places, a closely approximate estimate may legitimately be made. In length from above downwards the implantation cavity measures (in section 21.1.1) 3·67 mm., and in depth from before backwards 2·46 mm. It runs through 468 sections, each 10 microns thick, and therefore measures in breadth 4·68 mm.

Owing to the damage received by the specimen, much the same qualifications have to be made with regard to the measurements of the blastocyst as applied to the implantation cavity. The blastocyst, however, is not so much torn as the decidua capsularis, and accordingly a still more accurate approximation to exactitude can be made in regard to it. Exclusive of the thickness of the wall, and of the outgrowing villi, the blastocyst runs through 272 sections, each 10 microns thick, and therefore measures 2720 microns or 2·72 mm. from side to side. This is its greatest internal dimension. In section 22.1.5, which is the largest section of the blastocyst in which it is complete and untorn, the other two dimensions are respectively 1·66 and 0·84 mm. As this section is almost exactly through the middle of the blastocyst, it may be taken as giving the maximum dimensions with almost perfect accuracy.

On comparing it with the other youngest complete human ova, it is found to approximate fairly closely to that of Jung:—

Bryce-Teacher, $0{\cdot}77 \times 0{\cdot}63 \times 0{\cdot}52$.
[1] Linzenmeier, $0{\cdot}75 \times 0{\cdot}615 \times 0{\cdot}525$.
Peters, $1{\cdot}6 \times 0{\cdot}8 \times 0{\cdot}9$.
Fetzer, $1{\cdot}6 \times 0{\cdot}9$.
v. Spee, $1{\cdot}5 \times 2{\cdot}5$.
Herzog, $2{\cdot}32 \times 0{\cdot}8 \times 1{\cdot}2$.
Heine-Hofbauer, $2{\cdot}38 \times 0{\cdot}98 \times 1{\cdot}2$.
Jung, $2{\cdot}5 \times 2{\cdot}2 \times 1{\cdot}0$.
Johnstone, $2{\cdot}72 \times 1{\cdot}66 \times 0{\cdot}84$.

Decidua Vera.

The condition of the decidua vera was studied in the sections of a portion removed from the anterior wall of the

[1] Unfortunately, this paper was in the press before Linzenmeier's description of Stoeckel's ovum was published. The ovum was shown at the Gynæcological Congress at Munich in 1911. It was obtained, perfectly fresh, *in situ* in a uterus removed for repeated hæmorrhages one month after curettage. The ovum is, therefore, less than a month old. The uterus was almost certainly pathological. In its stage of development the ovum would appear to lie between the Bryce-Teacher ovum and Peters' ovum. The embryo consists of an amnion and yolk sac, the former measuring $0{\cdot}105 \times 0{\cdot}09$ mm., the latter half these figures. There is a definite embryonic disc measuring $0{\cdot}21 \times 0{\cdot}105 \times 0{\cdot}02$ mm. The embryonic anlagen are contained in a loose mass of mesoderm continuous with that lining the blastocyst, a distinct advance from the stage in the Bryce-Teacher ovum. The mesoderm is just beginning to form cores to the villi. The intervillous space is filled with blood, fresh and apparently in recent circulation. The two layers of trophoderm are well defined, but towards the periphery of the trophodermic shell seem to merge into each other indiscriminately. The implantation cavity is closed by a closing coagulum, lenticular in shape, consisting of partially organised blood and fibrin and trophoderm cells. The endometrium shows no true decidual cells, but is in a state of premenstrual congestion and œdema.

The basal blood spaces are partly capillaries and partly blood-filled glands. The surface epithelium was intact except over the closing coagulum. The illustrations are disappointing.

cavity—as far as possible from the site of the ovum. It measured 6·5 mm. in thickness. It presents in a characteristic way the appearance of two layers—the superficial compact and the spongy. It is crammed with decidual cells, which are most easily observed in the compact layer, although present in the trabeculæ between the glands of the spongy layer. No mitotic figures have been observed in the decidual cells, but this may well be due in part to the length of time that elapsed between the death of the patient and the fixation of the specimen, as well as to the somewhat crude fixative used.

The surface epithelium is almost completely wanting. Here and there in the depressions around the mouths of glands a few columnar cells are to be seen in series, and here and there a few isolated cells are to be found sticking up aslant like the last remnants of a derelict palisade. Probably this is due to maceration, for a similar condition prevails in the glands themselves. In them the epithelial cells are for the most part desquamated and lying loose in the lumina—the whole decidua having a catarrhal appearance. Indeed the gland spaces are so much filled up with epithelial debris that the differentiation into the spongy and compact layers is slightly obscured at a first glance.

The blood-vessels of the decidua vera are not unduly dilated. Here and there the veins are enlarged, but there is no great congestion, although the whole membrane is distinctly œdematous. Scattered throughout, but most marked in the proximity of the venules, there is a slight infiltration with round cells that look like lymphocytes.

Decidua Basalis.

The ovum is embedded in the superficial compact layer, and therefore lies upon a cushion of spongiosa and the deepest part of the compacta. This latter is, however, hardly recognisable

as such owing to the presence of large blood spaces. The differentiation into the layers is not very obvious in the basalis, owing to these blood spaces, and to the catarrhal condition of the glands.

Decidual cells are to be seen in large clumps or islands in the compacta near the border zone. Elsewhere they are almost completely absent, and broadly speaking they are not so marked a feature of the basalis as of the decidua vera.

Immediately posterior to the ovum there are a great many large blood spaces crammed with maternal blood corpuscles (Fig. 4). In most cases these spaces may be recognised as distended capillaries or venules, either by tracing vessels into them and observing their sudden widening out, or by the recognition of the flat endothelial lining. In some cases where this recognition is impossible the spaces may be true interstitial spaces. In the border zone, indeed, the whole stroma is infiltrated with blood. In one or two cases the presence of numerous degenerated epithelial cells in the blood suggests that the space was originally a gland space.

In the deeper portions of the decidua the congestion is less marked, and the normal corkscrew vessels are recognisable.

Throughout the entire decidua basalis, but most marked in the neighbourhood of the ovum, there is a definite leucocytic infiltration, in many cases considerable clumps of lymphocytes being seen in the proximity of a vessel.

The glands are very numerous and very dilated. The catarrhal condition of the epithelium has been referred to already. Towards the surface the glands appear to be pushed to the sides to make room for the blood spaces. They diverge and run up on either side into the capsularis, becoming more or less parallel to its surface.

Fig. 4.—Complete section of ovum and surrounding decidua, showing blood spaces opening into intervillous space.

BS—Blood spaces in decidua basalis.
DB—Decidua basalis.
DC—Decidua capsularis.
IS—Intervillous space.
M—Blastocyst wall (ruptured).
O—Blood spaces opening directly into intervillous space.

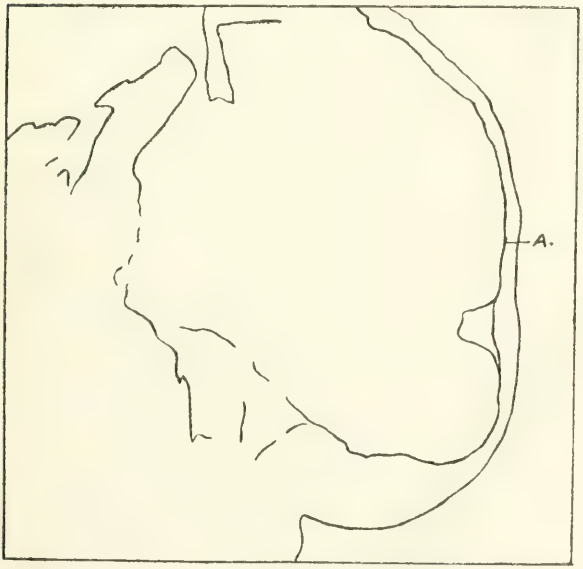

Fig. 5.—At A in the decidua capsularis gland tissue is present (*see* Fig. 6).

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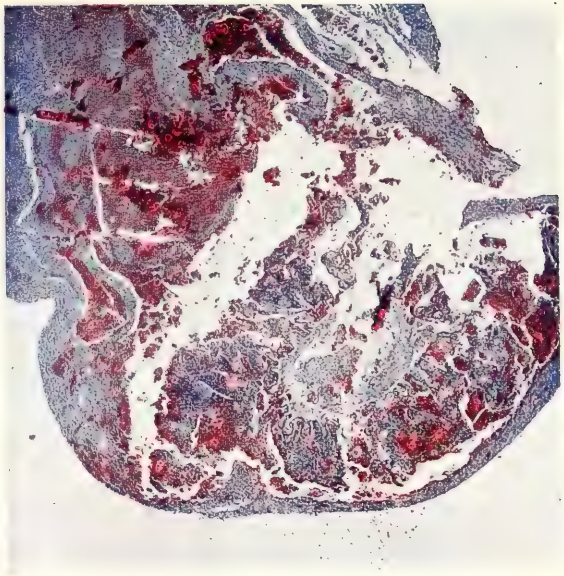

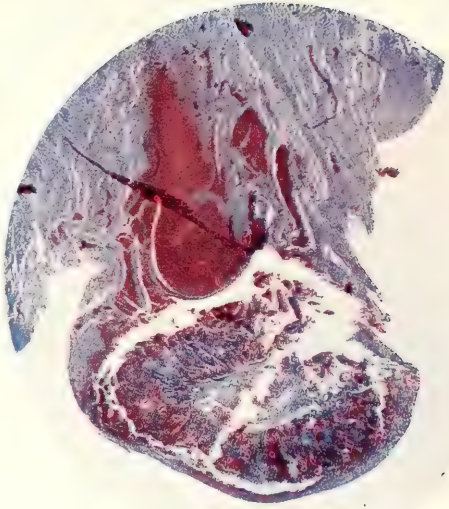

Decidua Capsularis.

One of the most noticeable features about the present specimen, and one which is obvious to the naked eye, is the marked extent to which the embedded ovum projects above the surface of the surrounding decidua. At the first glance it looked like a wart, and on closer examination like a pedunculated wart. This is borne out by the examination of the sections. The projection containing the ovum has a relatively narrow base, which it overhangs at the lower edge (Fig. 4).

It is convenient for purposes of description to follow Pfannenstiel in dividing the decidua capsularis into two divisions—the *equatorial* and the *polar*. By the equatorial portions are meant those parts which form a ring around the ovum and are directly and immediately continuous with the decidua basalis. By the polar region is meant that part which covers the summit of the embedded ovum and is farthest removed from the basalis.

In thickness the decidua capsularis gradually diminishes as we follow it from the equatorial to the polar region. Over the latter its average thickness is about 0·1 mm., while in the equatorial regions it attains as much as three times that thickness.

In the equatorial regions it consists of very much the same structures as the basalis—that is to say, of œdematous endometrial stroma infiltrated with blood, and containing many distended vessels full of blood. As has already been mentioned, the glands of the basalis are pushed aside by the wedge-shaped area of large distended blood spaces, and they run up into the capsularis for a short distance parallel to the surface. Disconnected portions of glandular tissue are, however, recognisable in the capsularis at a point very near the polar region (Figs. 5 and 6). I have not been able to recognise any gland opening into the implantation cavity.

The stroma of the capsularis does not contain any large decidual cells, but it is invaded (particularly on its inner aspect and in the neighbourhood of the attachment of the cell-columns) by trophodermic cells. The polar decidua consists very largely of fibrinoid material with a thin film of cells over its outer aspect. This fibrin is not altogether wanting on the inner aspect of the equatorial parts, but it becomes distinctly more prominent over the summit of the ovum. It is infiltrated on its inner side by trophodermic cells, and in one or two places little masses of syncytium have penetrated almost through its entire thickness. One or two of these syncytial masses actually have the striated border, the nature of which will be discussed later.

The Border Zone.

As has just been indicated, the border zone on its capsular aspect—or in other words the inner edge of the capsularis—consists of endometrial stroma infiltrated with trophodermic cells. Here and there a layer of fibrin is noticeable, particularly over the polar region, but this is neither universal nor continuous. In many places the cells of the capsularis are very chaotically disarranged, as if the tissue had been torn up by œdema, and many of them are more or less degenerated. There are numerous small spaces filled with blood, and in many cases these open directly into the implantation cavity.

The basal aspect of the implantation cavity is made up largely of the thin walls of the distended blood spaces. These walls are in some cases little thicker than films, composed of tissue which has undergone a fibrinoid degeneration and is invaded freely by trophodermic cells and plasmodium. In many areas this invasion has led to a loosening of the structure, which has permitted the escape of blood into the implantation cavity. In short, the implantation cavity is lined indiscriminately by invading trophodermic cells and plasmodium, by fibrinoid, and by more or less necrotic decidual stroma.

Fig. 6.—Decidua capsularis, showing remains of a gland. Below it is an elongated darkly stained mass of syncytium.

Fig. 7.—Section of small blood space in decidua basalis, showing degeneration of its wall into "symplasma maternum conjunctivum."

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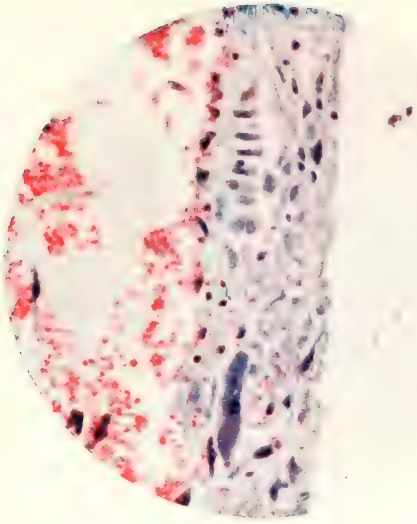

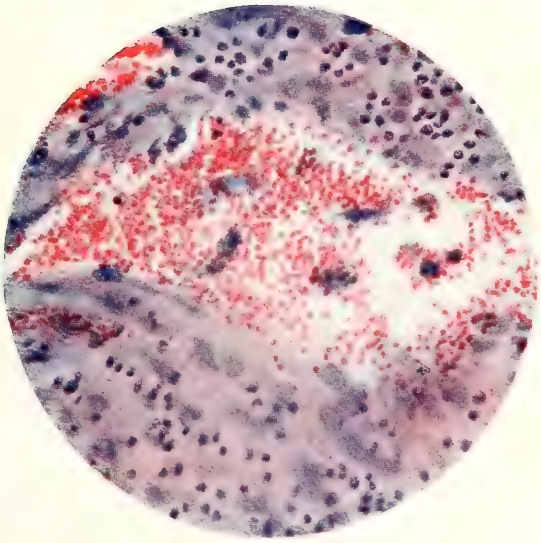

In at least two instances a distended blood-vessel may be seen opening directly into the implantation cavity (Fig. 4). In one place the wall of a blood space appears to have degenerated. The cells have lost their outline and the nuclei are swollen, elongated, and pale. The protoplasm contains brownish granules. Thus the tissue exactly corresponds in appearance and staining reactions to the syncytium of the outer part of the intervillous space (Fig. 7).

The Chorionic Villi and the Intervillous Space.

All round the circumference of the chorionic vesicle villous processes are observed growing out towards the surface of the decidua. These villi are, practically speaking, equally numerous and equally long all round the chorion—that is to say, there is not yet any increased exuberance of growth towards the decidua basalis, such as occurs later. The chorio-decidual space or intervillous space is full of maternal blood, which penetrates into all the interstices between the villi.

The proximal portions of all the villi are hollowed out by a mesodermic core. In many places this core has, unfortunately, been withdrawn inwards from its epithelial covering by the shrinkage of the mesoderm during fixation. Where it has thus been separated from its covering it presents a very smoothly defined edge, not at all unlike an endothelium. There is no appearance of embryonic capillaries in the villi. In addition to the core the villi have a double covering of trophodermic epithelium—an inner layer of cytotrophoderm and an outer layer of plasmoditrophoderm (Fig. 8).

The cytotrophoderm—Langhan's layer—is present mostly as a single layer over the surface of the chronionic membrane and the greater part of the villi. But towards the tips of the villi it becomes heaped together into large columns or masses. These unite with each other by irregular lateral outgrowths,

and spread themselves outwards to the surface of the implantation cavity in a shapeless and chaotic network. At the decidual surface they become attached by the trophoderm cells spreading laterally over the face of the decidua and also invading it (Fig. 9). The cells of the cytotrophoderm are rounded or oval, and contain rather deeply staining round nuclei which fill up the greater part of the cell.

The plasmoditrophoderm—the syncytium—is very irregular in its distribution. In some places it is absent over the surface of the chorionic membrane; in others it forms an almost unrecognisably thin layer; while in yet other parts it is very obvious as masses or buds, or as slender strands spreading over the surface of the cell columns, and stretching between them (Fig. 10). Farther out towards the decidua it is mostly found in such solid lumps or strands. On the surface of the chorionic membrane the syncytium where present has long, thin, flattened nuclei, diffusely stained. In the more uniform strands stretched over the cell columns and villi these oblong flat nuclei are still visible. But in the strands and clumps towards the periphery of the intervillous space the nuclei are more often larger, and rounded or oval in shape, vesicular, and faintly stained. Frequently these last nuclei are shrunken and crenated, and ghostly in appearance. The protoplasm of the syncytium is throughout foam-like and in many parts vacuolated. Everywhere this layer presents the appearance of tiny brownish granules, which are refractile, scattered through the protoplasm, and particularly numerous in the perinuclear protoplasm. Sometimes these granules are so numerous as to give a strand of syncytium the appearance of a brown streak along the middle of it.

In the outer part of the intervillous space there are numerous large free cells, some with large pale nuclei, others with numerous small dark nuclei. The former can usually be traced by serial sections to the strands with the large degenerate nuclei referred to above. The latter can also usually be traced to syncytial

FIG. 8.—Portion of chorionic membrane with outgrowing villi.

C—Mesodermic core of villus shrunk away from epithelium.
CH—Chorion.
T—Trophodermic cell column.
W—Space left by shrinkage of core.

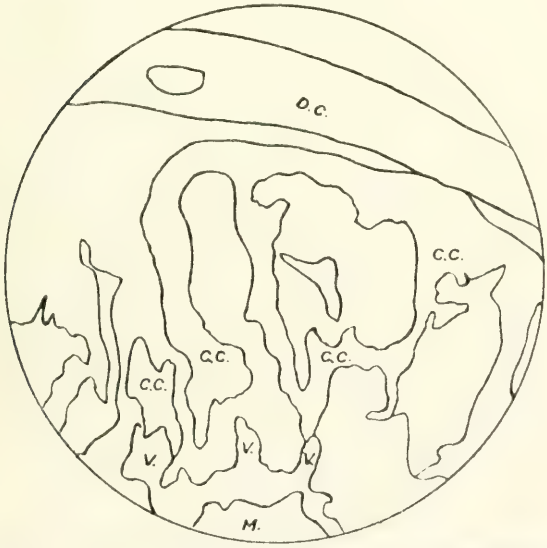

FIG. 9.—Showing attachment of trophodermic cell columns to decidua.

CC—Cell columns.
DC—Decidua capsularis.
V—Villus.
M—Mesoderm of chorion.

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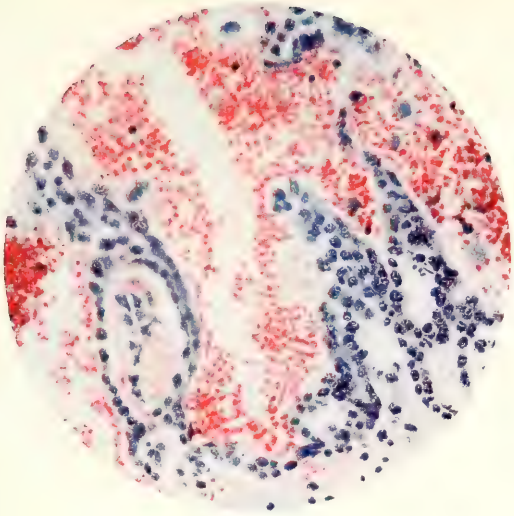

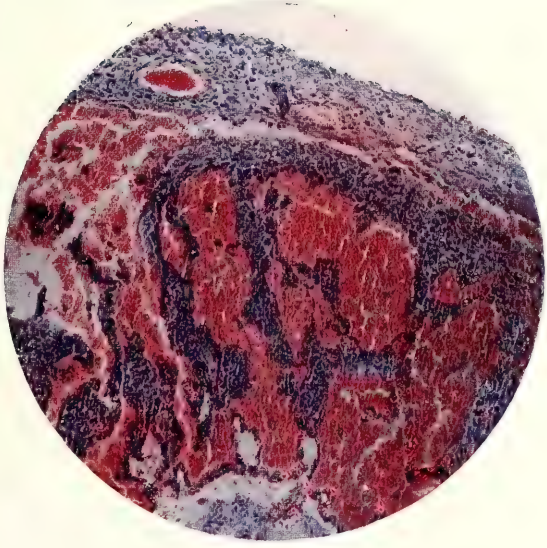

FIG. 10.—Trophodermic cell columns, with strands of syncytium (syn.).

FIG. 11.—Portions of trophodermic cell columns with empty spaces (A) which do not communicate with the intervillous space.

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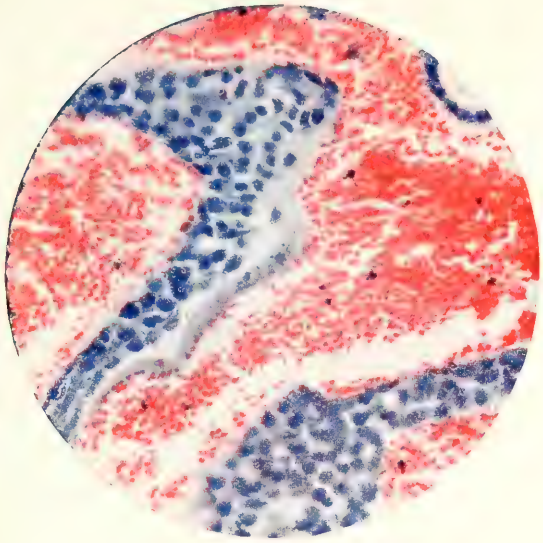
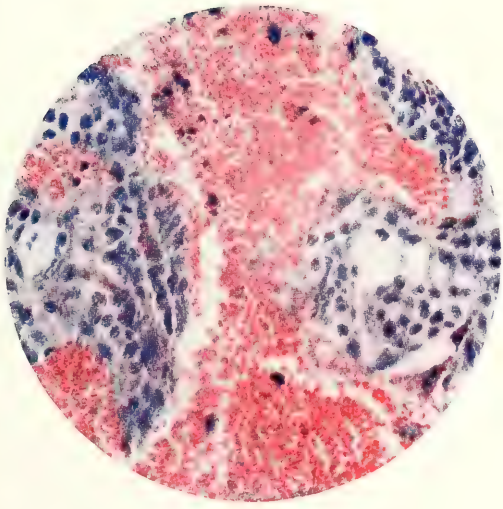
FIG. 10. — Tropidoneuric cell column, with strands of syntypium (type 1).

FIG. 11. — Portions of tropidoneuric cell column, with empty spaces (A) which do not communicate with the intercellular spaces.





buds, the nuclei on cross-section appearing smaller and round. Some of these wandering cells appear to be quite detached. In one or two instances the syncytium of these giant cells shows faint indications of a striated border.

There is a certain amount of vacuolation in the cell columns, empty spaces appearing, devoid of blood or any other content but a little structureless matter. These spaces are not due to the withdrawal of the mesodermic core from the villi, as they occur far beyond the utmost limits of the mesoderm in the substance of the cytotrophoderm columns. Serial sections do not show them to communicate with the intervillous space (Fig. 11).

The Chorionic Membrane.

The epithelium has already been described in connection with the villi. Both epithelial layers stain deeply with hæmatoxylin and eosin, giving the ovum a dark outline even under low magnification.

The mesoderm of the chorion constitutes a fairly uniform layer of about 0·05 mm. in thickness. It consists of a delicate connective tissue with elongated spindle-shaped cells having round or oval nuclei arranged mostly parallel to the surface. The inner portion is less nucleated and gradually becomes continuous, with a structureless debris inside the vesicle. This debris or "magma" does not fill up the entire vesicle, but is quite irregular in its distribution. The outer edge of the mesoderm where it is shrunk away from the epithelium has a smooth appearance not unlike an endothelium.

Embryonic Anlagen.

Owing to the damage received by the specimen the embryonic rudiments are considerably folded and at one or two points broken. It is therefore impossible to identify them with

absolute certainty, and I shall in the first place merely describe what is to be seen.

As one follows through the sections one comes first upon a vesicle which is somewhat crumpled, but is roughly triangular in shape (Figs. 12 and 14). This vesicle passes through thirty-five sections, and therefore measures in this direction 0·35 mm. Its other maximum diameters are 0·39 and 0·21 mm. In most

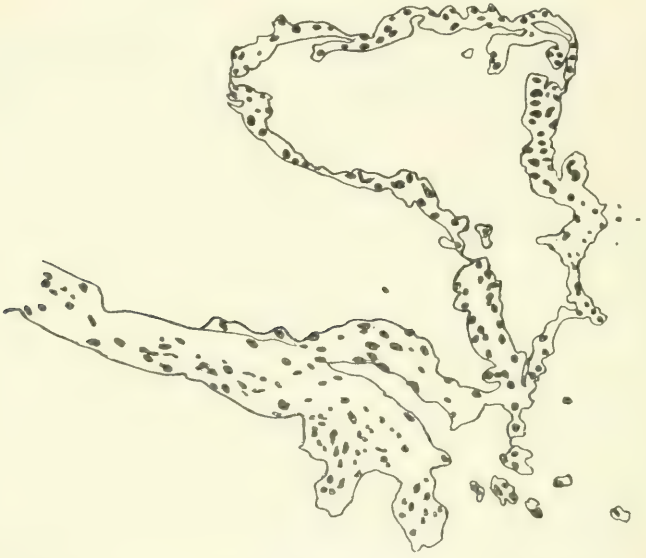


Fig. 14.—First vesicle and its attachment to the mesoderm of the blastocyst wall.

parts it presents two definite layers of cells. The outer layer, which is sometimes very much thinned, contains cells with oval nuclei. In the thinned parts these are flattened out and arranged parallel to the surface. This layer corresponds in every way to the mesodermic lining of the chorion, and with this tissue it is at one angle of the triangle definitely and substantially continuous. The inner layer is composed of rather larger cells which stain more deeply, and contain large

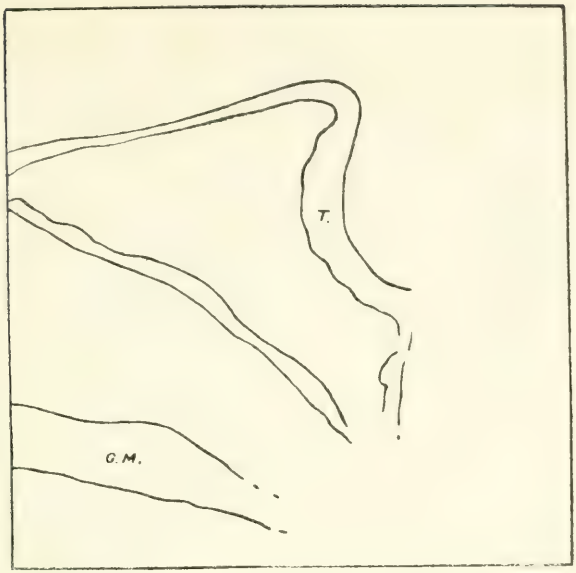


FIG. 12.—First vesicle of embryo.

CM—Chorionic mesoderm.
T—Thickening which *may* represent an embryonic area.

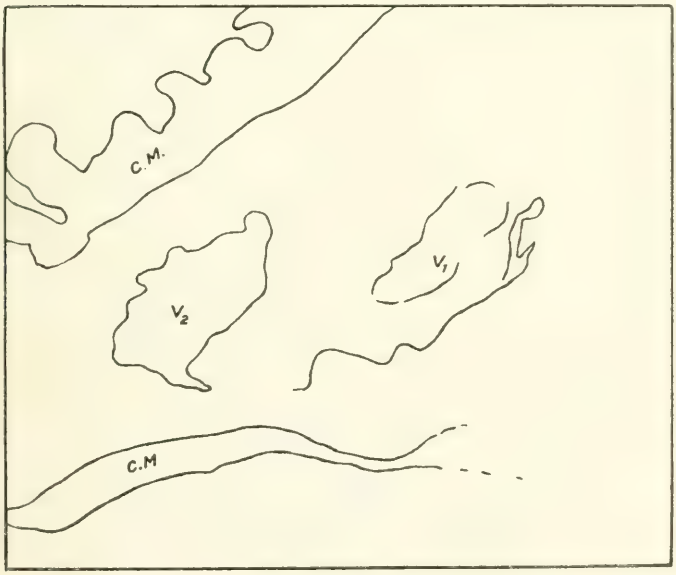


FIG. 13.—Both embryonic vesicles.

CM—Mesoderm of chorion. | V_1—First vesicle.
V_2—Second vesicle.

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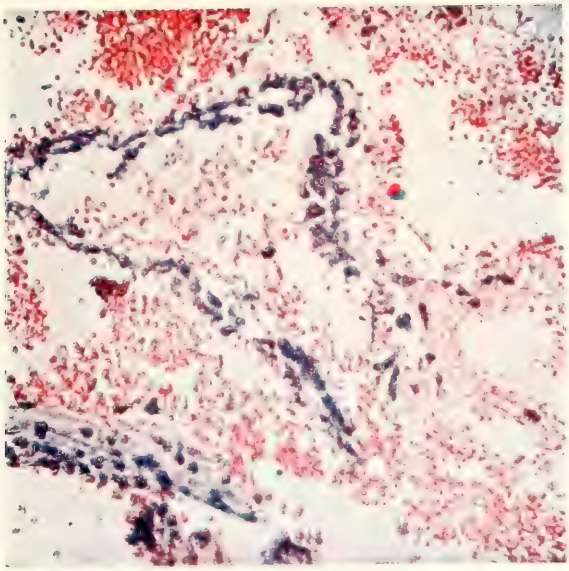
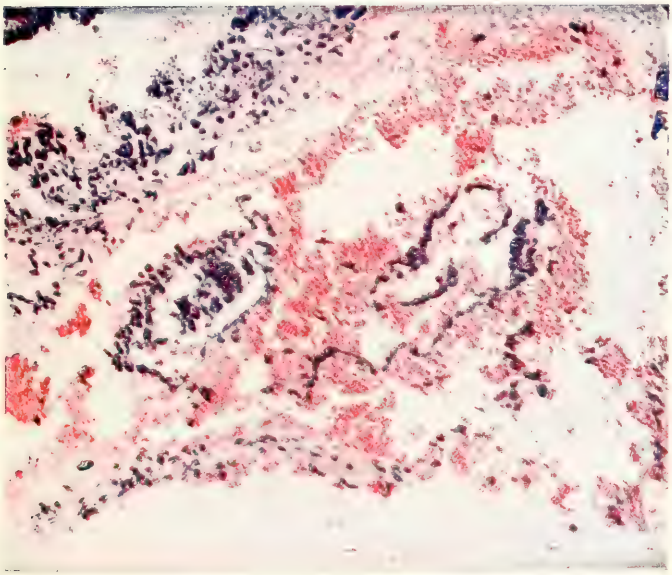
Fig. 12.—First version of embryo.
(M—Cheiloneurus incongruens.
P—The bearing which may represent an embryonic area.

Fig. 13.—Third embryonic condition.
(M—Mesoderm of clinging ... ?—Larval vesicle.
V—Second version.





round or oval nuclei with well-marked, deeply staining chromatin network.

The shortest side of this triangular vesicle presents an

Fig. 15.—First vesicle with pocket at lower right corner. B—Blastocyst wall.

appearance very like an embryonic area—that is to say, it is composed of cells heaped together in three or more irregular rows. In this area, which measures 0·21 by 0·2 mm., the nuclei

are more oval, and tend to be arranged perpendicularly to the edge of the vesicle.

At one end of this "area" a considerable fold or pocket is given off, which, but for serial sectioning, might easily have been mistaken for a second smaller vesicle. It is, however, directly continuous with the larger portion of the vesicle (Fig. 15).

On the wall of the larger portion there are one or two thickenings which resemble the anlagen of blood-vessels. But

Fig. 16.—Portion of blastocyst with both vesicles. Compare Fig. 13.

on tracing these through several sections they are found to be merely localised thickenings, which it is impossible to denote definitely as the precursors of vessels. This vesicle is unfortunately ruptured at one end, and there are a few maternal red blood corpuscles present within it, as well as some of the mesodermic magma.

In the later sections containing this first vesicle a second vesicle makes its appearance to the left of the first (Figs. 13, 16, 17). This passes through twenty-six sections, and accordingly measures in this direction 0·26 mm. Its other maximum diameters measure 0·28 and 0·14 mm. It is also somewhat crumpled, and is roughly triangular in shape. In structure it is a replica of the first, presenting the same two layers of cells. At the angle farthest away from the first vesicle it also is

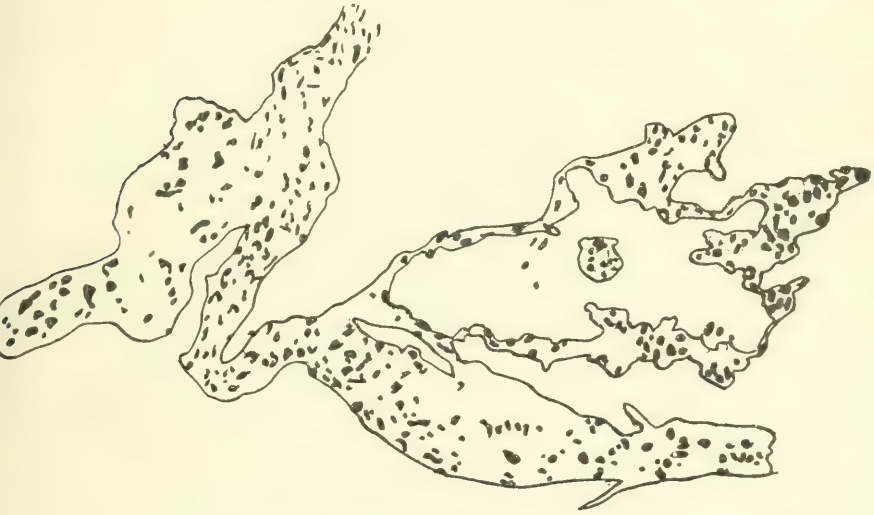


Fig. 17.—Showing attachment of second vesicle to blastocyst wall.

definitely and substantially attached to the blastocyst wall, its outer layer being for some distance continuous with the mesodermal lining of the chorion (Fig. 17).

In a number of the sections about one-third of the way through this vesicle, the cavity of the vesicle is almost wholly filled up with cells (Figs. 13 and 18). This flooring of cells appears to divide it more or less completely into two smaller vesicles. This vesicle, like the first, is not absolutely intact,

and several red blood corpuscles may be seen in its interior. At no point do the two vesicles come into actual contact, and

Fig. 18.—Portion of blastocyst with both vesicles, showing the flooring of cells in the second vesicle.

their attachments to the wall of the blastocyst are towards the opposite extremes of the one side of that structure.

The Corpus Luteum (Fig. 2).

Exact measurements of the corpus luteum have not been made, but the figure shows a section through it as nearly as may be through the middle. This section measures 1·3 by 1 centimetre. The normal shape is encroached upon by a small follicular cyst. The centre of the corpus luteum is occupied by a mass of pale finely granular material like mucus, stained pink with eosin. In it are several clumps and strings of red and white corpuscles. At the margins it becomes continuous with masses of red blood cells which fill up all the bays between the infoldings of the lutein layer. This layer is of varying depth, but never less than eight or ten cells deep. It appears crenated owing to the papillary ingrowths, each of which has a stout "core" of young and extremely vascular connective tissue. These connective tissue ingrowths appear to be branching in parts and pushing the lutein layer before them. There is a great deal of vacuolation amongst the lutein cells, some considerably large spaces being formed. The cells are polygonal or star-shaped, and their terminal fibrils stretch across these spaces. The nuclei are about twice the size of a red blood corpuscle, and stain well. The whole layer is very vascular, many capillaries being recognisable, and there is also a good deal of blood lying free in the stroma. At parts this blood is clearly continuous with that in the centre of the corpus luteum.

Second Specimen.

The second specimen I owe to the kindness of Dr William Fordyce. It was obtained from a private patient of his upon whom I assisted him to operate. She had a retroverted uterus, and an enlarged tender ovary on the left side. The abdomen was opened in order to perform a Gilliam's operation, and to

deal with the ovary if necessary. The left ovary was found to be slightly cystic, and to our surprise there was a blood-clot about the size of a large hazel nut protruding from the fimbriated end of the left tube (Fig. 19). The opposite ovary presented a recent corpus luteum on the surface. The left tube and ovary were removed, and the round ligaments shortened.

There was nothing in the patient's history to suggest, before the operation, the likelihood of a pregnancy, tubal or otherwise.

The tube and ovary were at once placed in Pick's (No. 1) solution and fixed. In the belief that the specimen was one of a tubal abortion, and that probably the ovum was broken up, I opened the blood-clot after three days, with the intention of making a museum preparation. Somewhat to my surprise I found it to contain an apparently complete chorionic vesicle, although no trace of an embryo was to be seen by the naked eye. The vesicle seemed to be entirely surrounded by blood-clot, but was entangled in or attached to the fimbriæ anteriorly and below. I then cut away the infundibular end of the tube with the blood-clot, and had serial sections made of the entire clot. The sections were cut 7 microns thick, and the plane of section vertical and parallel to the long axis of the tube—this being the plane of my first incision. The sections thus pass from the back to the front of the specimen. In all some six hundred sections were cut. Most of them were stained with hæmatoxylin and eosin, but some with Mallory's, some with van Gieson's, and some with Weigert's stains

Study of the sections revealed an intact chorionic vesicle, containing an early embryo, superficially embedded in the fimbriæ on the antero-inferior margin of the tube. The embryo is somewhat damaged, probably owing to the inevitable handling of the specimen during its removal, and partly to the unfortunate opening of the specimen before it was cut (Fig. 20).

Fig. 19.—Section through tubal ovum.

B—Blastocyst (chorion).
BS—Blood spaces in fimbriæ.
CM—Capsular membrane.
F—Fimbriæ of tube.
IS—Intervillous space, with sections of villi.
T—Mass of trophodermic cells.
YS—Yolk sac (ruptured).

Fig. 20.—Capsular membrane, with trophodermic cells applied to the inner (right) surface.

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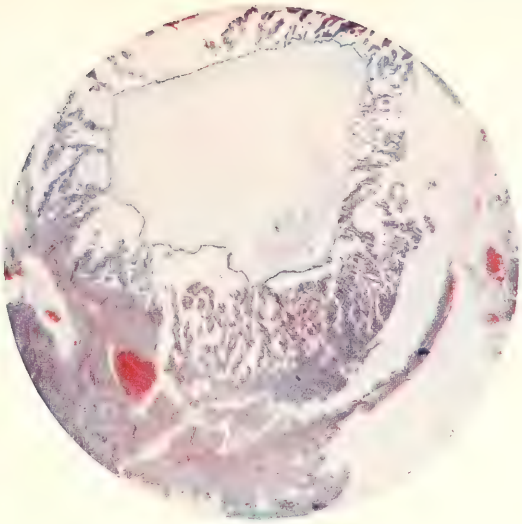

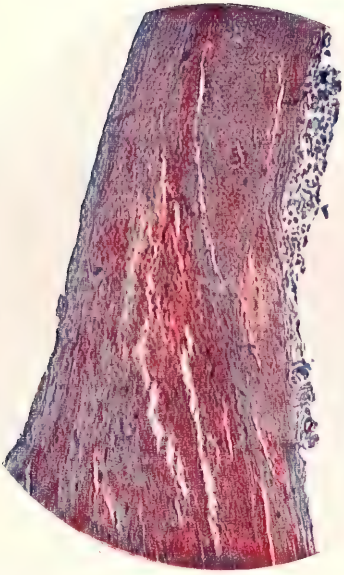

The rest of the tube showed no traces, either macroscopic or microscopic, of having been the seat of a gestation, or of any other abnormality.

Dimensions of the Ovum.

The chorionic vesicle, exclusive of its villi, runs through about 390 sections. Owing to the fact that the specimen was opened with a razor before the sections were made, some small portion of the thickness appears to have been lost—not more than the thickness of one or two sections. But a close approximation would give its measure in this diameter as 2·73 mm. In one of the middle sections (52.1.1.) its other diameters measure 6 and 5·6 mm. respectively. The whole vesicle is therefore of a flattened, almost circular shape. The flattening is in the diameter at right angles to the attachment to the fimbriæ of the tube, and in this way it corresponds to the lenticular shape of young ova embedded in the uterine decidua.

The embryo, including the two vesicles, is cut very obliquely, and runs through 306 sections. It therefore measures 2·1 mm. in this direction. The embryonic area proper runs through 230 sections, and thus measures 1·6 mm.

Attachments of the Ovum.

On the lower and anterior aspects the blood-clot was seen to be attached to the fimbriæ of the tube. Microscopic section shows that there is a definite organic attachment, thus indicating that the implantation was in all probability originally an infundibular one, and that we are not dealing with a tubal abortion, as at first I was led to suppose. The portions of the fimbriæ which come into the sections are seen to be covered, on those aspects not in contact with the ovum, with intact columnar epithelium, ciliated in many parts. On the one side, however, the epithelium is absent, the ovum being definitely but superficially attached to it.

On this latter aspect there is a shallow saucer-shaped depression, to the surface of which the ovum has attached itself, and which represents, therefore, the implantation cavity. At the edges of this "basalis" area, if we may so call it, there has evidently been an attempt at the formation of a "capsular membrane," and some of the elements of the tube wall can be seen stretching a short distance round the ovum. Nowhere does the stroma of the fimbriæ show any real decidual reaction in the shape of decidual cells, but there is in parts some enlargement of both muscle and connective-tissue cells.

The whole stroma, moreover, is in a most chaotic state of disarrangement. It appears to have been torn up by œdema, the cells lying at some distance from each other, and many of the cells are distinctly degenerated. The blood-vessels of the fimbriæ are enormously distended and full of blood. Several of them form large blood spaces which are separated from the intervillous space only by a very thin wall. The stroma is in many parts densely infiltrated with red blood corpuscles.

The capsular membrane consists of stroma elements only for a very short distance on each side. For the most part these elements are strongly reinforced by fibrinoid material, which forms the sole covering over more than half the surface of the ovum (Fig. 21).

Border Zone.

In relation to the capsular membrane just described there is little to correspond to the border zone of a normal implantation beyond the presence of the fibrinoid material and the occasional invasion of it by trophodermic islands, either cellular or syncytial. In the "basalis" area, however, in which there is maternal tissue present, the changes are closely similar to those in an intra-uterine implantation. Mention has already been made of the enlargement of some of the muscular and connective-tissue cells, as well as of the

FIG. 21.—Chorionic villus of twenty days' ovum.

Cap—Capillary being formed in mesoderm.
CC—Trophodermic cell columns.
S—Syncytium.
V—Villus branch.
VT—Villus trunk.
CM—Chorionic membrane.
F—Fimbria.
IS—Intervillous space.

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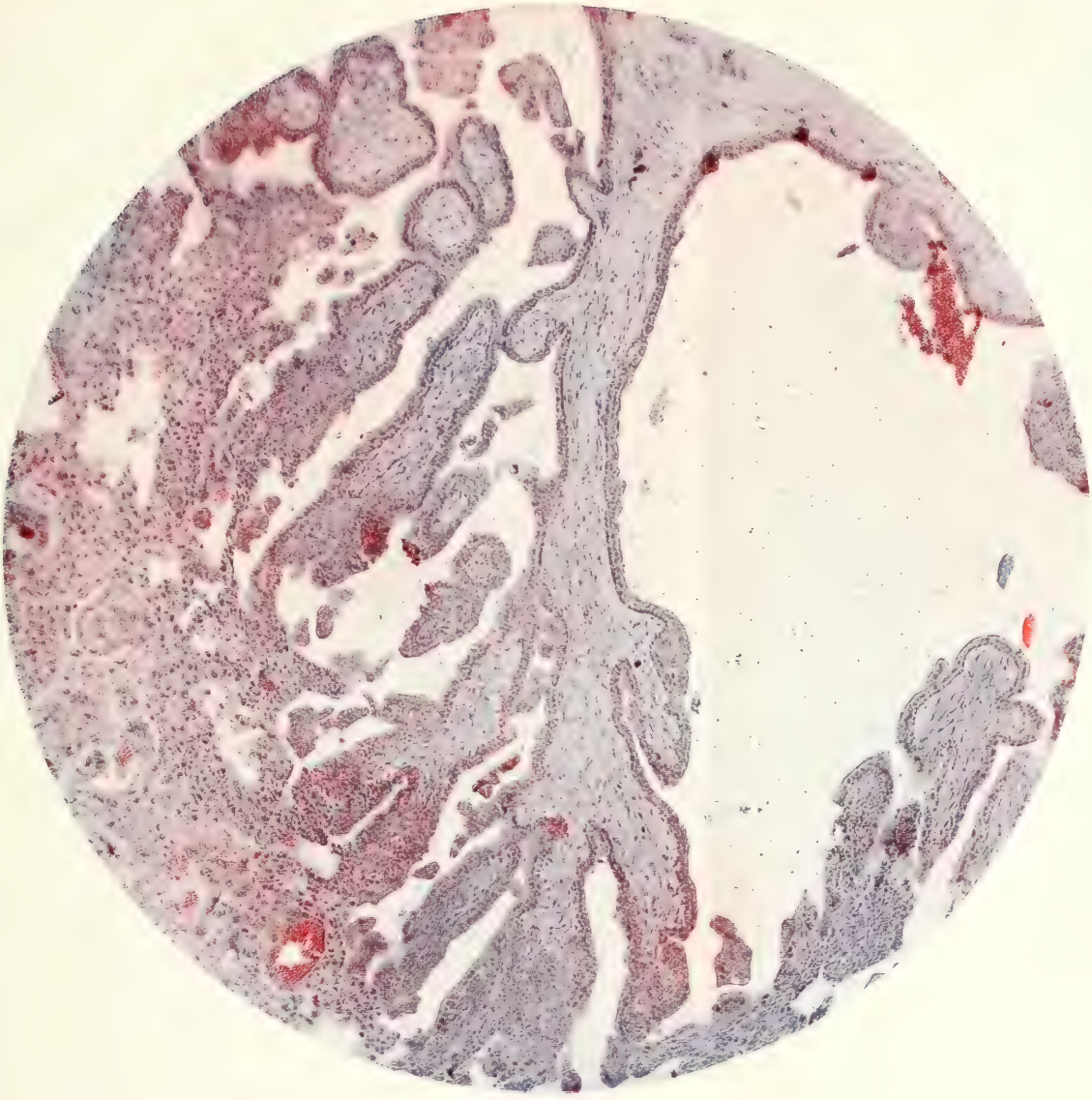


dilatation of the blood-vessels and the formation of blood spaces. In addition to this there is a very obvious invasion of the trophodermic elements into the tubal wall, both cells and syncytial masses being found at some little distance from the surface. There is also presumable evidence of the destructive action of the trophoderm in the presence of fibrinoid or semi-fibrinoid degenerated layers.

The Intervillous Space and the Chorionic Vesicle.

The intervillous space is not full of blood except in some portions. But throughout there is clear evidence of its having contained blood in the presence of occasional blood islands, of red blood corpuscles adherent to the epithelium of the villi, and in the blood which fills many of the smallest interstices. The villi are distinctly more numerous, more luxuriant, and more than twice as long on the side of the chorionic vesicle apposed to the fimbriæ of the tube. On the fimbrial aspect one villus measures 1·75 mm. in length, while on the opposite side of the blastocyst the average length does not exceed 0·75 mm. In other words, there is here a commencement of the differentiation of the chorion frondosum and the chorion læve.

In the chorion frondosum the villi are normal in their structure. I have been fortunate in getting one typical villus in longitudinal section through almost its whole length (Fig. 22). In this area also there is a great development and multiplication of trophodermic cells These form large cell columns which branch outwards from the ends of the shorter villi, and, uniting with each other, form a chaotic network. Where they reach the surface of the tube wall or the capsular membrane they spread out laterally, and to a marked extent invade these structures. This appearance is much more marked on the fimbrial aspect of the ovum

than on the capsular. On the latter many of the short villi end abruptly with no outgrowth of trophoderm. There are numerous spaces in these cell columns, some containing blood, others empty. Some of the empty spaces cannot be traced to the periphery of the columns, and are therefore enclosed spaces exactly similar to those described in the early intra-uterine ovum. Covering these cells columns there are strands and masses of plasmoditrophoderm or syncytium, to which reference will be made later.

The blastocyst consists of a layer of mesoderm covered by two layers of epithelium. All three layers are continued into the villi. The inner layer of trophoderm epithelium is Langhans' layer, and consists of a very regular single row of cells with rounded or oval nuclei which fill up the greater part of the cells and stain deeply. It is noticeable that where these cells pass into the cell columns at the ends of the villi as just described, they become distinctly enlarged.

In the chorionic membrane and the villi the syncytium is spread as a thin covering of fairly uniform thickness over the Langhans' layer. The protoplasm has a finely foamy appearance. The nuclei are mostly small and oval with a well-marked chromatin network, and deeply stained nucleoli. A few of the nuclei are larger and pale, but I have not observed any so ghostly in appearance as many seen in the earlier intra-uterine specimen. In some of the more tangential sections Langhans' cells appear in two or three layers, as also the nuclei of the syncytium. I believe, however, that this is largely due to the obliquity of the sections.

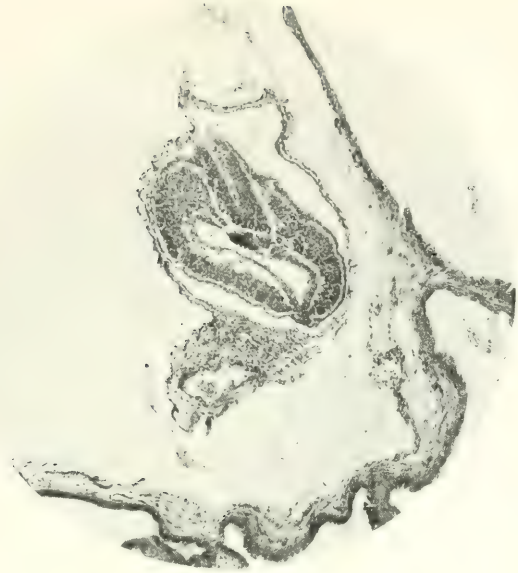
In the intervillous space syncytium is present, as mentioned, in the form of strands and masses on the outside, and not infrequently in the inside, of the trophoderm cell columns. In several places there are islets of multinucleated syncytium lying free. Some of these may be traced by serial sections to buds of the villous coverings; others cannot, and appear to be absolutely free.

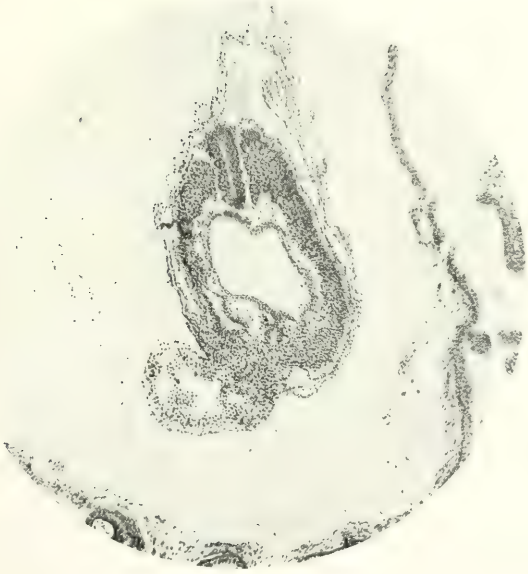
Fig. 22.—Embryo—hind sections.

A—Amniotic cavity. | C—Chorion.
BS—Belly stalk. | G—Gut.
M—Medullary groove.

Fig. 23.—Allantois (All.) just visible, passing from hind gut into the belly stalk.

[*Face page 74.*





An interesting point about the syncytium in this specimen is that in many portions—in practically all the buds and islands just described and in quite a number of the villi—it presents a distinct cuticle which has a striated appearance.

Inside the trophoderm the blastocyst is lined with a layer of delicate mesoderm, varying in thickness. This passes out into the villi forming their cores. Strangely enough, the structure of this mesoderm is more definite in the villus cores than in the portion lining the chorionic membrane. In one or two of the cores capillary vessels may be seen in the process of formation. In none of them was any blood observed.

The structure of the mesoderm is similar to that of the earlier specimen. The nuclei are more or less oval, and towards the outside are arranged parallel to the surface. Internally it fades irregularly into a structureless magma which is present here and there within the vesicle. At the corner of the vesicle corresponding to the greatest development of the villi, the mesoderm reticulum is rather greater in quantity, and here is to be found the embryo.

The Embryo.

The yolk sac has been ruptured, and part of its wall displaced outward. Including this displaced portion the whole embryo runs through 306 sections, and therefore measures 2·142 mm. Exclusive of this, the embryonic structures are present in 230 sections, which corresponds to 1·61 mm.—a more accurate measurement than the former. It has been cut very obliquely in a dorso-ventral plane. The hind portion is well seen, but more anteriorly the sections are not so satisfactory. In great part this is due to imperfect technique in mounting the sections, but the result has been to obscure some interesting appearances, and to render it difficult to be certain as to the absolute normality of the embryo. All that can be said is that those parts which can be well seen are normal.

In the earlier sections we find the caudal end of the embryo. The medullary groove, the notochord, the mesodermic somites, and the hind gut are all clearly visible (Fig. 23). Closely surrounding the whole is the amnion, consisting of two layers of cells. Posteriorly the hind gut dips down to the surface, from which it is separated by an anal plate or membrane. Below the embryo the amnion is attached to a mass of mesoderm—the belly stalk. This contains one large patent vessel with definite walls, and one or two small capillaries. The large vessel contains a number of red blood corpuscles, the great majority of which are definitely nucleated. In a small minority the nuclei have not taken on the stain satisfactorily. More anteriorly the gut becomes widened out, and the dip down to the anal plate disappears (Figs. 24 and 25). The belly stalk appears in direct continuity with the mesoderm of the body wall, the folds of the amnion coming down to the sides of the stalk. In the belly stalk the allantois may be distinguished as a slender column of epithelial cells running from the gut into the stalk. A little more anteriorly the gut is seen directly continuous with the widely opened yolk sac. The cœlom is just beginning to appear. The medullary groove is closed (Fig. 26).

At this point we come upon some unsatisfactory sections, but in more anterior sections we see the gut separated off from the yolk sac which is opened out widely and ruptured (Fig. 27). On each side the cœlom is visible, and projecting into the two halves are the anlagen of the heart. The amnion at this level is closely applied to the surface of the embryo. The yolk sac consists of two layers, much thinned out. At parts there are thickenings which seem to be the anlagen of vessels. The most anterior sections are, unfortunately, damaged.

Comparing these appearances with the data in the *Normentafeln* it is legitimate to conclude that the embryo comes in

FIG. 24.

A—Amniotic cavity.
All—Allantois.
C—Chorion.
G—Gut.
M—Medullary groove.

FIG. 25.

C—Cœlom.
M—Medullary groove.

[*Face page* 76.

Fig. 24.

A.—Auricular cavity. C.—Clavicle.
Au.—Auricle. O.—Os.
M.—Medullary groove.

Fig. 25.

C.—Coelom. M.—Medullary groove.

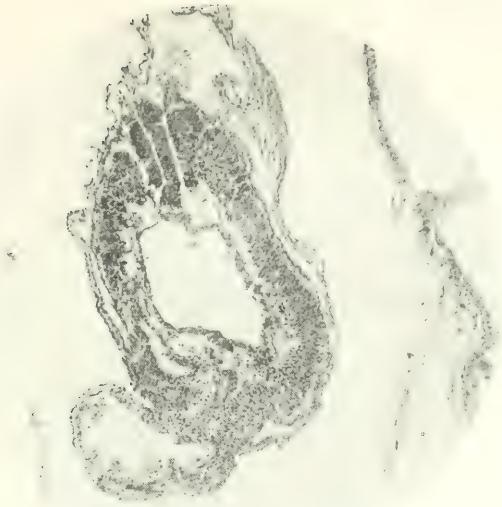


Fig. 26.

A—Amnion.
Coel.—Cœlom.
G—Gut.
H—Heart.
M—Medullary groove.
YS—Yolk sac, ruptured and opened out.

Fig. 27.—Syncytial mass, showing the striated border.

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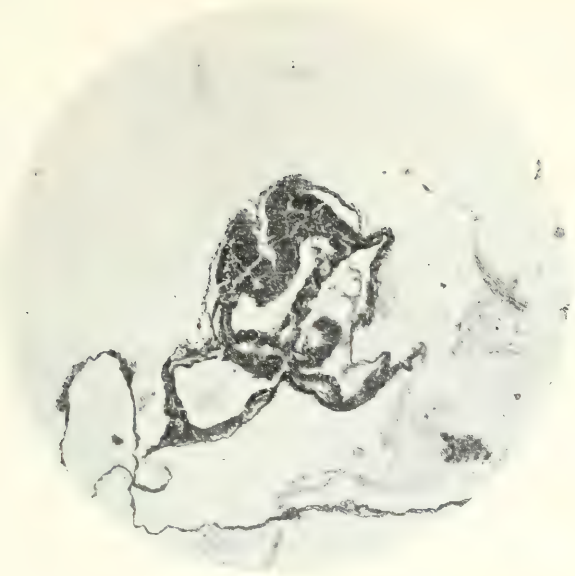

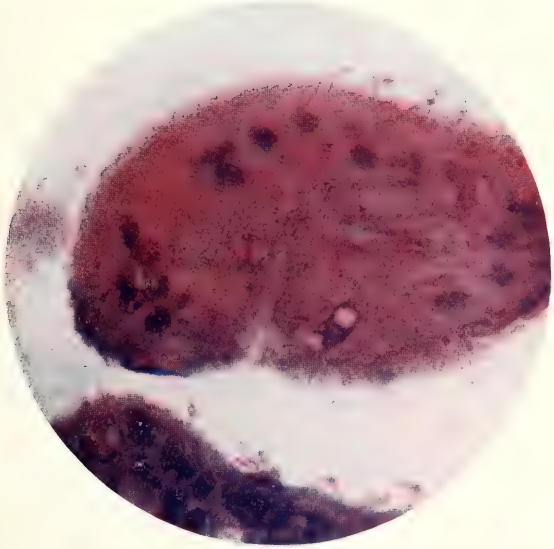

between the "Gle" embryo of v. Spee and the "Klb" embryo of Kroemer and Pfannenstiel.

Discussion.

The Decidua.—The main features of the so-called "decidual reaction" are the transformation of the embryonic connective-tissue corpuscles of the stroma—this origin can no longer be reasonably doubted—into the large decidual cells; the increase and distension of the glands, which become more tortuous in their course; the increased thickness of the endometrium, which becomes thrown into folds on the surface; and occasionally the congestion of the vessels and the œdematous nature of the stroma. What the stimulus is which gives rise to this profound change is unknown, as also the time of its occurrence—whether before or after the implantation of the ovum. Much speculation has been devoted to the question whether the stimulus may be due in some obscure biochemical fashion to the act of fertilisation, or to the influence of the corpus luteum, or to the actual invasion of the mucosa by the developing ovum. On none of these points have we any certain knowledge, but there are one or two facts from which probable conclusions may be drawn.

It is well known, for example, that analogous changes on a less marked scale take place in the endometrium during the premenstrual phase. It is also probable that a considerable number, if not the majority, of fertilised ova are embedded during that stage. Bryce and Teacher have argued strongly that the ovum *may* be embedded during any phase of the menstrual cycle, and this is in all likelihood the case. At the same time the premenstrual stage would appear to be the one in which most ova with a definite history were implanted.

In the second place, it is noteworthy that in several of the recorded specimens of early ova the decidual reaction has been

very imperfect. Thus the differentiation into the compact and spongy layers, which depends upon the increased growth of the glands, is present in Peters' case only in the immediate neighbourhood of the ovum, and in the older Heine-Hofbauer specimen it is wanting altogether. Again, in both Peters' and Jung's ova there was no clear formation of decidual cells, but only a few enlarged cells in the vicinity of the ovum.

Furthermore, we know that in the mouse, and more particularly in the guinea-pig, the endometrium undergoes no change whatever until the ovum has actually eaten its way through the surface epithelium.

Putting these facts together, it seems not unfair to conclude that in all probability the decidual reaction proper does not occur until the ovum begins to invade the endometrium, and that the slight changes present in some recorded cases are merely the persistence of premenstrual conditions of varying intensity. The rapidity with which the reaction ensues may quite naturally vary in different cases.

It must not be forgotten that a strong argument against this provisional conclusion appears to rest in the formation of a uterine decidua in cases of extra-uterine pregnancy. But there is no evidence, so far as I am aware, to indicate that the uterine change in such cases occurs before the ovum has embedded itself in its abnormal situation, and therefore we may legitimately conclude that the change is again due to some biochemical influence consequent upon the invasion of the ovum.

This argument practically assumes the truth of the view, first promulgated by Sir William Turner, that the decidual reaction is a protective one on the part of the maternal tissues. Everything points to the correctness of this view. There is, for example, the effort at decidual formation in the tube itself during pregnancy in that situation, as well as the appearance of decidual cells in the ovary, peritoneum, and cervix in some

non-pregnant conditions. In all cases, moreover, the appearance of decidual cells is accompanied by a leucocytic infiltration, as has recently been pointed out by Meyer, and therefore it may be regarded as in some measure comparable to the ordinary reactions of inflammation. If the biochemical stimulus be blood-borne from the trophoderm, as seems most plausible, the uterine reaction in tubal pregnancy may easily be explained as a praiseworthy but misplaced effort on the part of the maternal tissues to counteract it.

At the same time the decidual reaction is in all probability indirectly but equally protective to the embryo, in that, by supporting and strengthening the maternal capillaries, it prevents a too sudden and too extensive opening up of these vessels—the probable consequence of which would be to tear up the delicate attachments of the ovum. The formation of a decidua is therefore to the mutual advantage of both mother and child.

My first specimen shows a very characteristic decidual reaction. The differentiation into the *compacta* and the *spongiosa* is well marked, although it is somewhat obscured by the free desquamation of the epithelial cells lining the glands. Decidual cells are found throughout in the compact layer, and to a lesser extent in the trabeculæ of the spongy part. I failed to find any mitotic figures in the cells, but this may be due to the circumstances in which the specimen was obtained. As a rule, such changes are only to be seen in perfectly fresh and carefully fixed preparations. Marchand, Bonnet, and others have recorded the presence of many. On the other hand, Herzog, whose specimen was obtained and prepared in an almost ideal manner, failed to detect any karyokinetic changes.

The comparative absence of the superficial epithelium in the present specimen is, I believe, largely due to maceration. It is absent in the Bryce-Teacher specimen, as might be expected

after its adventures. It is wanting also to a great extent in Cova's ovum, in spite of good preservation. In the great majority of other cases, however, it is present and in fairly good condition.

This maceration, which is evident also in the deeper parts of the glands in my ovum, makes it impossible to recognise the papillary projections of the epithelium into the lumina of the glands, which are by some regarded as a characteristic feature of the early decidua.

Apart from this maceration the glands seem to have suffered but little change even in the decidua basalis. They have obviously been pushed aside by the expansion of the ovum, the wedge-shaped space between them being occupied by the "cushion" of dilated blood spaces. Bryce and Teacher suggest that the glands are more resistant to the destructive action of the trophoderm than the other tissues, and this may well be so, although they ultimately suffer some dissolution. Frassi has shown conclusively that the glands may be opened into laterally, and it is probable that this may be effected either by the eroding action of trophodermic buds, or by the digestive action of the proteolytic ferments of the trophoderm, while that tissue itself is at some little distance.

This is presumably the explanation of three observed facts —firstly, the presence of epithelial "rests" in the "border zone," formerly but probably erroneously regarded as relics of the superficial epithelium; secondly, the opening of glands into the intervillous space, as observed by Frassi and Fetzer—the glands as they run up towards the equatorial portion of the decidua capsularis being opened into laterally; and thirdly, as a consequence of this the presence of blood in the gland spaces, which has been noted by Heukelom von Siegenbeek, Leopold, and Fetzer amongst others.

These conditions I have not observed in the present specimen. The blood spaces nowhere have, unequivocally, the characters of glands, but for the most part appear to be dilated

vessels. The flat endothelial lining can be seen in many places, while in others the identity can be established by tracing fully formed vessels into the spaces, and noticing how these vessels suddenly widen out into sinuses. Indeed they are absolutely comparable to the blood spaces in the second (tubal) ovum, where there is no question as to the possibility of their being glands.

In at least two places the blood spaces can be seen opening into the implantation cavity or intervillous space. At these points there is no indication of any proliferation of the endothelium over the surface of the implantation cavity. Nor is there any definite or continuous lining of that space by syncytium. On the contrary, the space is lined somewhat indiscriminately by fibrinoid tissue and trophodermic elements.

The œdematous condition of the decidual stroma is noted in most early ova, but it is usually associated with a certain amount of congestion of the blood-vessels throughout the whole decidua. Jung refers particularly to a condition of venous hyperæmia. In my specimen there is no marked congestion in the decidua vera, but merely some enlarged veins here and there. There is in the decidua basalis, especially around the border zone, a noticeable escape of red blood corpuscles into the stroma. This condition is more widespread in my specimen than in Jung's, where it was confined to the immediate neighbourhood of the border zone. I have not detected anything that could be interpreted as the formation of new capillaries in this zone. This phenomenon is described by Peters, particularly in the capsularis. White blood cells, or at least what appear to be lymphocytes, are to be found in the stroma, particularly around the vessels, not only in the decidua basalis but in the vera and capsularis.

The Embedding of the Uterine Ovum.—In connection with the decidua capsularis I have already mentioned that the embedded ovum projects to a quite unusual degree above the

surface of the surrounding decidua. The only other specimens which resemble it in this respect are those of Leopold, Cova, Marchand, and Rossi Doria—particularly the last-named. This circumstance had led Cova to suppose that the ovum was necessarily arrested by a projecting piece of the endometrium, upon which it proceeded to engraft itself. Rossi Doria, on the other hand, finds in it support for the older view of the process of embedding, namely, that the ovum comes to rest on the surface of the mucous membrane and is gradually enclosed by an upgrowth of decidua capsularis around it. He modifies it, however, and states his belief that the implantation is effected by a combined method—the ovum eroding its way through the superficial epithelium into the substance of the mucous membrane, while at the same time the surface decidua grows up round it as a true "capsularis."

In the present specimen the projection of the ovum is even more marked than in Rossi Doria's specimen, and, as has been said, gives it almost the appearance of being pedunculated. At the same time I do not consider that there is any need to explain this by any peculiarity in the mode of implantation. Cova's view can neither be substantiated nor denied. It remains a mere pious opinion. On the other hand, there is no evidence to support Rossi Doria's view. There is, it is true, no mushroom-like stopper of fibrinoid material ("gewel spilz") at the point of entrance, as in Peters' and some other cases; but there is clear evidence that the polar portion of the decidua capsularis consists practically entirely of fibrinoid material—the natural interpretation of which is that it represents the stretched out "closing coagulum" at the point of entrance of the ovum. Moreover, there is absolutely nothing to indicate any active growth of the decidual elements in the decidua capsularis, such as one would expect if it were actively growing up around the ovum. There is, for example, no sign of cell division.

I believe it to be more likely that there was originally a closing coagulum at the point of entrance, but that it has disappeared partly by absorption and partly by stretching. Furthermore, the projection of the ovum is probably to be explained by two things—firstly, that the ovum may have been rather superficially embedded; and secondly, that, as a result, the direction of least resistance, in which growth would naturally take place, was, even more markedly than usual, towards the surface.

Another interesting point in the present specimen, which at the same time goes to prove that the capsularis is no new formation, is the presence of relics of glandular structure in it well up towards the polar region. This is clearly shown in the photographs (Figs. 5 and 6).

Embedding of the Tubal Ovum.—At this stage it is interesting to compare and contrast with the conditions in the early uterine pregnancy those of the second specimen—the early tubal pregnancy.

The trend of a good deal of the recent work upon tubal gestation has been to throw the responsibility for this abnormality more upon the ovum than upon the tube. The old idea of preceding disease of the tube is only rarely borne out by an examination of the affected tube. In a great many cases the tube is remarkably healthy. This is so in the present instance, the rest of the tube showing no abnormality nor trace of disease.

When we look to the ovum for an explanation, we are forced to conclude that in some circumstances it must be fertilised at or near the ovary, and that its development goes on so rapidly, or it is so delayed in reaching the uterus, that it becomes provided with the necessary trophodermic apparatus for embedding itself while still in the tube. In the present instance there is a particularly beautiful illustration of the truth of this view. In the ovary removed along with the

pregnant tube there is no recent corpus luteum. On the ovary of the other side there was observed at the time of operation what we took without hesitation to be a recent corpus luteum. The ovum must therefore have travelled from the one side of the uterus to the other. Further, assuming that it was fertilised on the surface of the ovary from which it arose, and that the journey to the mouth of the opposite tube occupied several days, it is possible that by the time it arrived at this point it was ready to implant itself. Under these circumstances it is less surprising that it engrafted itself on the fimbriæ, instead of passing along to the ampullary portion of the tube, which is so much more common a site for implantation.

It must be admitted that the assumptions thus made are in the present state of our knowledge not susceptible of proof. We have no actual knowledge of the frequency or rarity of the presence of spermatozoa in the peritoneal cavity, although the possibility of it is proved by cases of ovarian and primary abdominal pregnancy. Again, we have no proof that the journey from the one side of the uterus to the other must occupy the ovum for several days. Lastly, we have no certain knowledge of what, in the case of man, brings the ovum to rest either in the tube or the uterus, or of the condition of the developing ovum prior to the blastocyst stage. As a matter of fact, these considerations are rather lost sight of in all theories of tubal gestation which seek to place the whole blame upon the tube.

It is legitimate to conclude that when the human ovum escapes from the ruptured Graafian follicle it is surrounded by the *zona pellucida*, and outside that by some of the immediately adjacent cells of the *discus proligerus*, the so-called *corona radiata*. Through these two coverings the spermatozoon penetrates, and development begins. The subsequent fate, as well as the function, of the corona radiata is unknown. In his

Hunterian Lectures Professor Robinson has suggested that its cells may provide pabulum for the growing ovum while the latter is still in the higher reaches of the Fallopian tube. On the other hand, it may go to form an albuminous coating, such as is found in the ova of several animals—the dog and the rabbit, for example. This layer by its stickiness probably acts as a means of fixing the ovum to the mucous membrane. We do not know, however, that any such layer is formed in the human ovum, and apart altogether from it we are still in the dark as to how long the zona pellucida persists. In some animals—Tupaja javanica according to Hubrecht—it disappears at the two-celled stage. In the mouse and rat it persists to about the eight-celled stage, and even later in the bat. In the mole and the shrew it persists until the embryonic ectoderm comes to the surface of the ovum, while in the dog and the ferret it does not disappear until after the appearance of the primitive streak and the commencement of the formation of the mesoderm. Generally speaking, it disappears early in those animals in which the ovum is early embedded in the decidua.

The functions of the zona are somewhat obscure. In all probability, according to Robinson, it is not so much a protection from actual pressure as from premature attachment. Thus in animals in which the embryonic ectoderm comes to lie on the surface of the ovum the zona persists until the embryonic ectoderm cells have reached a stage of development at which they no longer tend to fuse with the decidua. On the other hand, in animals such as the mouse, rat, guinea-pig, hedgehog, and bat, in which the embryonic ectoderm never comes to the surface but remains separated from the maternal tissues by other cells or a fluid-filled space of considerable size, the zona disappears at an early stage. In these animals its function is probably just to prevent the union of the chorionic cells with the decidua until the ovum has attained

a sufficient size. To this second class there is reason to believe that man and the apes also belong.

Bearing in mind the possibilities lying behind our ignorance of these points it becomes apparent that in this case, and in a great many other cases of tubal pregnancy, the arrest of the ovum in the tube may be due to the premature or the excessive development of the hypothetical albuminous layer around the ovum, or to the premature disappearance of the zona pellucida. Of the conditions governing any of these circumstances we are totally ignorant.

But to return to the present instance, I believe that my assumptions, although they cannot be proved, afford the simplest explanation of the conditions found—namely, that the journey of the ovum from the right ovary to the mouth of the left tube—a journey considerably longer than that to the interior of the uterus by the ordinary route—occupied the ovum for several days, and that by the time it reached the tubal infundibulum it was ready, in a perfectly normal sense, to embed itself. Moreover, if this occurred in the case of a woman with tubes that were, so far as could be seen, perfectly healthy, it is probable that a similar explanation underlies quite a number of other cases of tubal pregnancy.

The actual process of embedding in the case of tubal pregnancy is now regarded as being as strictly analogous to what occurs in the uterus as the differing circumstances will allow. There is every reason to believe that the ovum eats its way through the superficial epithelium of the tubal mucosa exactly as it erodes its way into the endometrium. The first difference met with is the absence of anything like a thick stroma underneath the epithelium of the tubal mucosa. In most cases, therefore, the ovum eats its way right into the muscular wall of the tube, and forms there its implantation cavity. This is probably a slower process than sinking into the substance of the soft endometrium, and accordingly the

surface epithelium does not get the same chance of closing over it as the decidua capsularis has in the uterus. Thus the tissue which separates the ovum from the lumen of the tube degenerates almost as soon as it is formed, and is present in most specimens as a membrane composed largely of fibrin. To it is applied the name "capsular membrane." In some cases, however, the ovum sinks deeper, and several authors have demonstrated tubal muscle elements in the capsular membrane.

In many cases there are obvious attempts at the formation of a decidua. The relative absence of a connective-tissue stroma makes it impossible for this to be a marked feature in the tube, but even in the presence of such stroma the reaction is often absent, and if present is usually only in small isolated patches. The vascular changes in the immediate neighbourhood of the ovum, and the effects of the action of the trophoderm, are analogous to what is found in the case of uterine pregnancy.

The above applies in the main to ampullar pregnancies, which form the majority of tubal gestations. But it will be seen that, *mutatis mutandis*, it explains all the appearances of the present specimen.

Thus the saucer-shaped hollow in the fimbriæ, in which the ovum rests and to which it is attached, shows that the ovum had eroded its way through the surface epithelium into the substance of the fimbriæ. This portion of the fimbria or fimbriæ represents the decidua basalis, and is the seat of a marked reaction similar to what is found in the true decidua basalis. There are no decidual cells, but merely an enlargement of some of the muscle and stroma elements. The whole tissue is markedly œdematous, and there is a striking enlargement of the blood-vessels with the formation of very large blood spaces separated from the intervillous space only by thin films of tissue.

A capsular membrane has been formed, which in the "equatorial" parts presents the structure of the fimbriæ. Over more than half the ovum, however, this structure is replaced by blood-

clot woven through with coarse streaks of fibrin, and irregularly interspersed with cells. Most of the latter appear to be trophodermic in nature. Whether this capsular membrane originally consisted to a greater extent of fimbrial tissue which has subsequently undergone fibrinoid degeneration, or whether it has been from the outset largely made up of blood-clot, it is impossible to say. The appearances incline me to the latter view, and I think the large expanse of fibrinous clot must be regarded as corresponding to a closing coagulum at the point of original entrance of the ovum, which has been excessively stretched.

This in itself, however, is not a sufficient explanation, as it cannot be held to include the whole of the blood-clot that was seen at the time of operation, and is shown in the photograph. To cause this there must have been some bleeding, and although I have not been able to detect the source of the bleeding in the course of the microscopic examination, yet I think it must have come in some way from the surface of the equatorial portion of the capsular membrane. It is likely that the blood oozed out very slowly, and having percolated gradually round the ovum formed the clot. In doing so it probably reinforced the earlier fibrinoid capsule just referred to. Some such occurrence as this is at least possible considering the very shallow implantation of the ovum, and the fact that, as has been shown in my other specimen as well as by many other observers, the vessels of the decidua may be opened up by the influence of the chorionic elements at some little distance from the actual chorionic cells.

In this connection I gave full consideration to the possibility of the specimen being, as I thought probable at the first, one of a tubal abortion—that is to say, of an ovum which had been implanted farther along the tube, and had then been dislodged and expelled to the abdominal ostium without the embryo being killed, and which had then engrafted itself secondarily upon the fimbriæ. As already stated, however, the rest of the

tube appeared perfectly healthy, and in the parts examined microscopically there was not the faintest sign of any undue congestion, still less of any destruction of the lining epithelium. The normal outline and shape of the tube were unaltered. Further, the clot, on the outside at anyrate, appeared quite fresh and recent, and therefore must have been formed long after the ovum had attached itself to the fimbriæ. It is, of course, generally agreed that after such an abortion the tube very quickly returns to its normal condition, but I hardly think it could do so in quite such a short time as might have been available in this case, or to such a complete extent in that time. I believe, therefore, that I am right in regarding this as a primary infundibular pregnancy.

From the nature of the site of implantation there can be nothing to correspond to a decidua vera, and, as has been stated, there is practically no change in the rest of the tube. Even the vascular engorgement is very localised and does not extend beyond the affected fimbriæ. In this it resembles the intra-uterine specimen, where the engorgement was practically confined to the decidua basalis.

The surface epithelium present on the distal side of the fimbriæ shows no sign of enlargement or of degeneration or maceration. Even the cilia can be readily detected.

I have not detected any blood-vessels opening directly into the intervillous space.

The Border Zone.—The border zone, or transition zone or "Umlagerungszone," marks the division between the fœtal and maternal tissues. As might be expected it is not a sharp line, for it is in truth the fighting line where the conflict between the maternal cells and the invading trophoderm takes place, and it is strewn with such of the dead on both sides as have not already been carried off the field or otherwise disposed of. In the earlier stages there is even more evidence of the deadly nature of the conflict than in the later stages. The Bryce-

Teacher ovum, for example, has a very marked zone containing degenerated and necrotic cells of both maternal and fœtal origin. But in the later stages there is evidence that the maternal tissues are succeeding in limiting the invasion. Thus we do not find the large degenerated "maternal" cells described by Bryce and Teacher in the margin of the intervillous space.

Put briefly, the border zone in the first specimen consists of decidual stroma very much torn up by œdema or by the influence of the trophodermic elements, and invaded every here and there by these elements. This invasion is most noticeable where the cell columns of trophoderm are attached to the surface of the cavity. There is thus an irregular distribution of trophodermic cells all round the cavity, but no definite layer of syncytium such as is found later—the "basal ectoderm" of Langhans. Streaks and patches of fibrinoid material are also irregularly present all round the cavity, and no doubt represent Nitabuch's fibrin layer. This tissue is, however, not sufficiently regular in its distribution to warrant one in regarding it as a third layer separating maternal and fœtal elements, as is claimed by Fetzer.

Jung and Frassi have pointed out that the infiltration of the decidua with leucocytes enables one to differentiate between the fœtal and maternal tissues, because these cells are unable to penetrate the fœtal area. In my specimen there is a general infiltration by leucocytes through the decidua, and particularly in the neighbourhood of the ovum. At the same time their presence is not sufficiently marked to enable me to regard them as a satisfactory boundary line.

I have mentioned that the decidual tissue is in parts so opened out as to permit the escape of blood from the underlying blood spaces. I have also mentioned that the whole tissue is infiltrated with red blood corpuscles in the neighbourhood of the ovum. The result is that round the edge of the implanta-

tion cavity there are numerous tiny bays or pools of blood into which the red cells seem to be filtering through the decidual stroma. The edge of the cavity thus somewhat resembles the seashore at low tide, when there are many small pools communicating with the sea on the one side and receiving innumerable small runnels or streams of water from the other side.

There is also a clear connection on a larger scale between the blood in the implantation cavity and the maternal blood spaces. In two instances there can be seen opening into the cavity blood-vessels, which can be traced in the other direction into the large blood spaces of the decidua basalis. In this respect the specimen agrees with those of Frassi and Cova.

The tubal specimen agrees in the main with what has been said of the uterine one. The streaks of fibrin are on the whole more uniformly present. This is particularly so on the inside of the capsular membrane. At the same time trophodermic cells may be seen far beyond it, and it can in no sense be regarded as a dividing line between fœtal and maternal tissues.

A very interesting point of difference is that in the tubal specimen the invasion of trophodermic elements into the tube wall is distinctly more marked. Detached masses of both syncytium and Langhans' cells may be seen at quite a considerable distance from the edge of the gestation cavity, and, furthermore, the natural arrangement of the maternal tissues in the border zone is much more destroyed. The whole tissue in the zone is indeed ploughed up to a quite extraordinary extent.

It seems quite fair to assume that this is due to the absence of the decidual cells, and of the support and resistance which they lend to the maternal tissues. At anyrate, this is a feature that has been noted frequently in tubal gestations, where deportation of portions of syncytium along the blood stream is not an unusual occurrence.

The stroma of the tube wall in this zone is infiltrated with red blood cells, but there is not the same formation of small bays opening into the intervillous space, nor have I seen any vessels opening directly into the space. The leucocyte infiltration is a much less marked feature of this specimen.

The Intervillous Space.—In the uterine specimen the intervillous space is quite full of maternal blood. In the tubal specimen it is not so, but the space contains several considerable blood islands, as well as innumerable individual red corpuscles, clinging to the surface of the villi, and lurking in many of the smaller interstices. This forms clear evidence of its having been full of blood.

There can be little doubt that that is the normal condition, and that there must be a slow circulation by means of such channels as have been described. The force of the blood stream must be broken by the intervention of the large blood spaces in the decidua basalis, and the danger of a forcible escape of blood into the intervillous space during these days of slender attachments of the ovum greatly obviated. The arrangement is indeed an ideal one for securing a safe and slow circulation.

Several specimens have been described in which there was no blood in the intervillous space, and on the strength of such a specimen, otherwise apparently normal, Marchand actually stated the opinion that it was the normal condition. The presence of blood in the Peters' ovum, for example, he regarded as due to the effects of the caustic alkali poisoning from which the patient died. Marchand's own specimen, however, was obtained from a woman who died of severe internal hæmorrhage, and this may explain the absence of blood from the intervillous space. Since then Herzog's specimen, obtained under almost ideal conditions, has been found to have the space full of blood; while Frassi, whose ovum did not contain blood, regards that as an abnormal feature, due in all probability to the squeezing of the uterus during its removal by vaginal hysterectomy.

The fact that the blood, although outside the vessels, does not clot, has been pointed out again and again. It is the only instance in the body of such a phenomenon in connection with normal (non-menstrual) blood. The explanation would seem of necessity to lie in some attribute of the trophoderm elements—both the cellular and the plasmodial layers—which prevents clotting. Hofbauer has suggested that this may be of the nature of a fine layer of albumose secreted by the trophoderm.

The further contents of the implantation cavity are in both cases the villi, and the outgrowths of the trophodermic epithelium. In both specimens the epithelial covering of the chorionic membrane is very similar—a single layer of Langhans' cells regularly arranged, and outside that a thin layer of syncytium. In the tubal specimen, which is obviously older, there is a beginning differentiation into chorion læve and chorion frondosum. In one or two parts also the villi of the second specimen show the commencement of capillaries in the mesoderm c core.

In the first specimen all the villi, and in the second case the shorter ones which do not reach to the surface of the tube wall, terminate in great outgrowths of trophoderm. These cell columns branch very irregularly, uniting with each other laterally, and reaching out to the surface of the implantation cavity to which they attach themselves. At the points of attachment the cells invade the maternal tissues as has been described.

The "Third Layer."—Before passing on to the discussion of the trophoderm, a brief reference must be made to the question of the existence of the much-debated third layer between the two-layered trophoderm and the decidua. In some of the lower animals a series of giant cells has been described lying between the trophoderm proper and the maternal tissues. They first appear about the time that the embryonic mesoderm is formed. In the mouse and the hedgehog, for example, they are frankly

phagocytic, and portions of red blood corpuscles and decidual cells may be seen in their interior. Hubrecht accordingly called them *deciduofracts*, and they are regarded as a means of enlarging the implantation cavity.

In the mouse, to quote from Lochhead's admirable summary, "Duval stated that each was derived from a cell of the fœtal ectodermal wall of the yolk sac, and later from a cell of the ectoplacental cone. As they increase in number, they form a distinct layer between the yolk sac and the wall of the implantation cavity, and some wander into the decidua and lie singly or in groups. In their interior degenerating leucocytes are frequently seen. Sobotta also stated that they were fœtal in origin, and helped to fix the ovum and erode the maternal capillaries. More recently Kolster has brought forward evidence from their histological appearance that they are transformed decidual cells, and this is strongly supported by Disse's investigations on the field-mouse, in which the giant cells are found before the ovum has become embedded, and the first to appear are at an appreciable distance beneath the surface epithelium. A second series of smaller size appears later in the lumen and wall of the implantation cavity. Jenkinson also recognised two groups, but assigned to them different origins, fœtal in the 'Eikammer' and maternal in the decidua.

"All authorities agree that they are phagocytic. The tissue around them undergoes fatty degeneration, and in their interior may be seen remnants of connective tissue and endothelial cells and fat globules. Many capillaries are ruptured, and red and white blood corpuscles are also absorbed. Such an absorption of maternal tissue by the giant cells leads to an increase in the size of the implantation cavity, and a thinning of its wall. In spite of their abundant supply of nutriment, their life-history is short. No cell divisions occur, and they soon degenerate. Their contents are absorbed by the tropho-

blast, and their protoplasm shrinks to form a rim around the nucleus. Later still their remnants are also absorbed."

I have quoted this passage at length to show how dubious are the nature and origin of these cells even in lower animals, in which such a problem is much easier of solution than in man. Bryce and Teacher have described a number of somewhat similar cells in their human ovum, "lying free in the blood space within the necrotic layer of the decidua" in small excavations or bays. "Within the layer itself are seen spaces enclosing cells in different phases of degeneration whose nuclei are identical with those of the completely free cells, and the protoplasm stains the same dusky red in both cases." They admit that "these cells closely resemble bodies which are certainly cross-sections of plasmodial strands, but when traced through the sections it becomes quite certain that they are not continuous with the plasmodium, but are really isolated cells." Largely because they agree in nuclear characters and protoplasmic reactions with cells farther out in the decidua which are obviously degenerating maternal cells, the authors come to the conclusion that they are more likely maternal in origin. The cells are, they state, "quite different from the wandering trophoblast cells of later phases."

In Peters' ovum there are also some large cells, which the author compares to the deciduofracts of the hedgehog. Siegenbeek van Heukelom, too, describes such a layer, and regards its cells as fœtal, derivatives from the cytotrophoderm. "Whatever their origin," says Lochhead, "the mononuclear cells in man appear to be engaged in disintegrating mucosal tissue, and producing a zone of coagulation necrosis, *i.e.* a symplasma, around the trophoblast. But they differ from similarly situated cells in lower animals, *e.g.* the mouse, in showing no evidence of ingestion of formed tissue-elements."

The conclusion of the whole matter would therefore appear to be that it is a perfectly open question whether the cells

described in the early human ova are really comparable to the deciduofracts of the lower animals. My own specimens throw no light whatever on the problem. There are, as has been described, numerous wandering giant cells in the outer part of the trophodermic sheath and intervillous space, but these are all so closely similar to the cross-sections of plasmoditrophoderm strands, that their origin from these cannot be held in doubt, even although they are quite detached and isolated. It is, however, quite possible that such cells do occur in the earlier stages, such as the Bryce-Teacher ovum, and very soon thereafter disappear. As has been stated, they are very short-lived even in the lower animals.

The Trophoderm.— The cytotrophoderm cells (Langhans' layer) correspond closely in both ova. In the chorionic membrane they are arranged in a single row. This arrangement is continued over the trunks of the villi, but at the tips of such villi or their branches as do not reach the periphery of the intervillous space they become heaped up into the cell columns described. Where the arrangement is regular the cells are rounded, and the round or oval nuclei occupy most of the cell space; but in the cell columns they are so massed together that the shape becomes in many cases irregularly polygonal. The size of the cells also is greater in the columns, and where they spread out over the surface of the maternal tissues they have a superficial resemblance to decidual cells. Their direct continuity with Langhans' layer as well as a closer study of their appearance, however, make it quite clear that, as has also been shown by Williams, Berkeley, and Bonney, and a host of other observers, they are not maternal in origin.

In both specimens these cells invade the maternal tissues, and may be detected at some distance from the edge of the implantation cavity. This is particularly the case in the second specimen, and is probably as I have indicated, due to the absence of the decidual reaction.

A comparison of the syncytium, or more correctly the syncytiotrophoderm, in the two specimens reveals several very interesting points.

In the uterine gestation it is spread so thinly over parts of the chorionic membrane as to be hardly recognisable. On the outside of the villi it is more uniform in arrangement, and amongst the cell columns it is to be found in clumps and strands (Fig. 10). The protoplasm is always foam-like or finely granular, and more or less studded with fine brownish granules. In only one or two instances is there any suggestion of a striated border. Over the chorionic membrane and villi the nuclei are mostly long, flattened oblongs, staining diffusely. Elsewhere this type of nucleus is replaced in parts by large, pale, and often crenated and shrunken nuclei, containing one or two darkly staining nucleoli. Numerous apparently detached islands may be seen, some containing the large pale nuclei, others groups of the small, dark, round ones, probably the oval ones on section. In many cases these islands may be traced through serial sections to the strands mentioned. Others are quite detached, but there can be no reasonable doubt that their origin was the same.

In the tubal gestation the syncytium is more uniformly arranged over the outside of the entire chorionic membrane, and is always easily recognisable. On the outside of the villi it is more exuberant in growth, and the intervillous space contains a much greater number of buds and detached islands. As before, some of the islands or giant cells can be traced by serial section to villus buds, while others are entirely detached. The protoplasm is again very definitely foam-like, and stains uniformly with eosin, and apparently also with the hæmatoxylin, to judge from the purplish red hue. The nuclei are preponderatingly rather small and dark and are very numerous. A well-marked striated border is visible in many places. Over the chorionic membrane it is but seldom seen;

over the villi it is more frequent; whilst it is best and most constantly seen on the syncytial buds and the detached giant cells. Nowhere is there any appearance of the brown granules so uniformly present in the other specimen.

Of all tissues in the whole realm of histology the syncytium has almost certainly been the most discussed during the last twenty-five years. Peters, whose classic description of his ovum threw the first definite light upon it, mentions some ten distinct theories as to its nature and origin! Since that publication it has been demonstrated with a gradually increasing clearness that the human ovum is embedded in much the same way as in the case of the guinea-pig, that is to say, interstitially in the uterine mucosa. This knowledge at once threw out of court at least two of the more prominent views of its origin—namely, from the surface epithelium of the endometrium, and from the glandular epithelium. A further and still more conclusive proof of the erroneousness of these views has been obtained more recently in the numerous carefully described cases of ovarian pregnancy, in which the syncytium is found to be identically the same as in an ordinary intra-uterine pregnancy.

This leaves only two views of importance, one that it arises from the endothelium of the vessels of the decidua, and the other that it is fœtal in origin. The former view was last championed by Pfannenstiel, all other observers having abandoned it in favour of the latter. Recently, indeed, Frassi has given it the *coup de grâce* by showing from Pfannenstiel's own figures that what that observer regarded as endothelium may with much greater probability be looked on as an epithelial rest—that is, a portion of epithelium remaining in a gland which has been opened into laterally by the trophoderm and converted into a blood space. This occurrence has been referred to already in connection with the nature of the blood spaces in the decidua basalis.

In one area my first ovum shows an appearance which might readily have been taken to support Pfannenstiel's view—namely, a blood space lined with "syncytium" (Fig. 7). The cells have lost their outlines and the nuclei are elongated and pale. The protoplasm contains the brownish granules which are found throughout the syncytium in this specimen. I believe, however, that this tissue is not syncytium in the sense of plasmoditrophoderm, but rather a degeneration of the maternal tissues lining the space. In other words, it corresponds to what, as I mention below, Jung describes as "symplasma maternum conjunctivum."

The fœtal origin of the syncytium is indicated in a much more positive way in such an ovum as Bryce and Teacher's. In it there is no connection between the syncytium and the maternal tissues, whereas it is in intimate connection with the blastocyst wall.

While, then, it is generally agreed nowadays that the syncytium is the outer layer of the trophoderm, we have no exact knowledge of the earlier stages of its development or of its essential nature. Peters regarded it as the reaction of the cytotrophoderm to contact with the maternal blood, and both he and Jung have described appearances which led them to think that it is formed out of the cellular layer underlying it. The only appearance in my specimens which might be taken as supporting this view, is the presence of syncytial islands in the centre of some of the cell columns. This appearance can, however, also be explained by the fusion of two parallel cell columns, and the consequent enclosure of the syncytium between them.

Hubrecht and Bonnet regard the syncytium as an indication of intense functional activity on the part of the trophoderm when it comes into contact with the maternal structures. This implies an organic reaction of a more or less automatic kind between the fœtal and maternal tissues, just as Peters' theory

does. Whether or not this be the case, the term "contact" is certainly too restrictive. For there are large stretches of syncytium that never come into contact with the maternal tissues—such, for example, as the layer covering the chorionic membrane in between the villi. It is true that in some cases, as in my first specimen, the syncytium is irregular in its distribution over that area, but still it is present to some extent, and in many other specimens it forms a complete covering. Even in the earliest ovum — the Bryce-Teacher specimen—there is much syncytium that is not, and probably never was, in contact with the maternal tissues. Accordingly the stimulus to the reaction, if such it be, must be more far-reaching than actual contact.

From the study of my own specimens I am not inclined to regard the syncytium in the sense of a reaction of the trophoderm cells to contact or even association with the maternal tissues or blood. Such a reaction would be universal. But we find cytotrophodermic elements in intimate contact with the maternal tissues and blood without their being changed into a syncytium. On the other hand, the syncytium has everywhere, except in the degenerate forms, the appearance of being an intensely active tissue, designed primarily for the dissolution of the maternal tissues, and possibly later for the less aggressive functions of selecting and transmitting nutriment. I would therefore subscribe to the view that the syncytium is probably the outer layer of the trophoderm from the very outset. This does not, of course, exclude the possibility of its being formed continuously from the cytotrophoderm. Such an origin is most probable, but it is not specifically called out by association with the maternal tissues. Apart from this question of its origin, Grosser's view as to its changing functions, which will be mentioned in a later paragraph, seems the one which best suits our rather conflicting knowledge of the tissue.

Before elaborating this latter view, however, it is necessary

to consider and contrast the syncytium as present in such an early specimen as the Bryce-Teacher ovum and as present in older ova.

The Bryce-Teacher ovum is surrounded by a loose spongework of syncytiotrophoderm, which "forms an extraordinarily extensive spun-out investment for the ovum. It occurs in masses, bands, or threads. The nuclei are invariably small, and stain darkly." Peters' ovum represents a stage subsequent to this. In it the syncytium is distributed around the blastocyst in an extremely irregular fashion, but the "spongework" has largely disappeared. In the still older ova the arrangement becomes increasingly uniform as a layer on the outside of the cytotrophoderm, and the only trace of the previous arrangement is to be found in the irregular strands and masses interspersed among the cell columns towards the periphery of the intervillous space.

Some of these latter masses are regarded by Jung as degenerated portions of the syncytium or of maternal tissues, as the case may be. These he terms symplasmata—either symplasma syncytiale fœtale or symplasma maternum conjunctivum, according to their apparent origin. Another point of possible distinction between the earlier and the later syncytium is that the latter shows a striated border in many cases.

It will be seen that my first specimen corresponds to Jung's description. The masses and strands of syncytium containing large shrunken nuclei are what he calls symplasma syncytiale fœtale, and the fact that in my specimen these nuclei are pale and ghost-like, while in his they stain diffusely, is probably due to a more advanced degeneration. One instance of the symplasma maternum I have already referred to (Fig. 7). In the second specimen there is very little of this symplasma formation. On the other hand, the striated border is well marked in it—both possibly points indicative of a more advanced stage of development.

Grosser makes what seems a helpful and thoughtful suggestion when he speaks of there being, "so to speak," two generations of syncytium. "The first is associated with the implantation and with the intense histolysis which leads to the formation of the cavity of the egg capsule; it is represented by the syncytial shell of the Bryce-Teacher ovum." As time goes on this early syncytium may be supposed to degenerate into the symplasma syncytiale, which has all the appearance of a degeneration product. Meantime the histolytic function is largely usurped by the cytotrophoderm, which is seen invading the maternal structures to a much greater extent than the syncytium. The newer generation of syncytium, on the other hand, may be considered to assume either a secretory or an absorptive function, of which the striated border is probably an indication. This supposition involves the constant formation of new syncytium, and this can most easily be accounted for by the view that it is formed continuously from the cytotrophoderm. The appearances noted by Peters, Leopold, and Jung suggesting an actual stage of transmutation, confirm this. At the same time it is probable that the syncytium has in itself powers of reproduction, for syncytial buds may be seen in the placenta long after the cells of Langhans' layer have ceased to exist. Still, the disappearance of the cellular layer does largely coincide with the time when the demand for the formation of syncytium in large quantity begins to diminish, and accordingly the cellular layer may well be regarded as to a great extent, although not exclusively, the mother-layer of the syncytium.

In this connection I should like to point out that, even assuming the truth of this view, there may be other reasons associated with the relatively early disappearance of Langhans' layer in the placental villi. Lochhead has indicated one possible theory, which is suggestive. He points out that during the early weeks of development, in which both coverings are

present over the villi, the different organs of the fœtus, indeed the different tissues, are hardly sufficiently developed to be able to conduct their own metabolic activities, even on a limited scale. More selective discretion must therefore be exercised for them—in other words, there must be a more rigid filtering and selection of the substances passed through the villus epithelium to the fœtal blood. In the later months, on the other hand, the organs become increasingly more and more able to select their own particular forms of nourishment, and to carry on their own individual metabolism. The efficiency of the placental filter may therefore be relaxed. This stage coincides with the gradual disappearance of Langhans' layer.

The striated border of the syncytium (Figs. 28 and 29) has been studied mainly by Bonnet, Marchand, and Lenhossek. It has been described as a row of prickle processes—the Bürstenbesatz—and as a ciliated margin. Lenhossek studied it in a fresh specimen and maintains that the processes are not motile, but that they are provided with basal granules in the cuticle, which may be observed in specimens stained with iron hæmatoxylin. No one else has described such granules, and I have entirely failed to see them even in specimens stained with iron hæmatoxylin. The striated margin has, however, been described in quite a number of the young ova, including those of Jung and Herzog. Peters is not definite on the point, but speaks of "a delicate and thin, strongly refractive deposit, slightly frayed at the edges, on the surface of the syncytium." Rossi Doria regards it merely as a deposit of broken-up blood corpuscles on the surface of the syncytium.

Peters' description applies closely to many areas in my tubal ovum, and in regard to Rossi Doria's view it must be admitted that there is frequently a deposit which is quite recognisably made up of broken-up blood cells. But in many other parts the appearance is more definite, and, moreover, I have seen it in syncytium buried in the substance of the decidua

capsularis, where it would be impossible to imagine an accidental deposit of broken-down blood cells.

The tubal specimen affords the opportunity of contrasting in one and the same section this striated cuticle and the ciliated surface of the tubal mucosa, which is still quite obvious on the outer side of the fimbriæ. The comparison shows that the tubal cilia are more slender, as might be expected, since they are motile; whereas the "rods" of the striated border appear stiffer and straighter, and do not suggest the possibility of movement. In a word, they are more rod-like than the cilia themselves.

The question arises whether in the absence of any power of movement these processes are really rods, or whether the appearance may not be due to the presence of pores out of which secretion is passing. Against their being *merely* pores is their extraordinary regularity, as well as the straight, stiff appearance mentioned. At the same time it is difficult to conceive of their being unassociated with some function either of secretion or absorption, and accordingly it is quite possible that the spaces in between them represent pores.

Apart from such functional reason for their existence, it might be conceivable to regard them as a phylogenetic memory corresponding to the ciliated epithelial covering of some elementary marine forms of life. But this is harking back to a very remote ancestry indeed, and against it must be placed the fact that, so far as our knowledge goes, this appearance is not found in the earliest known syncytium (as in the Bryce-Teacher ovum), but only in the somewhat later stages.

The appearance of small refractive brownish granules scattered throughout the protoplasm, and more particularly the perinuclear protoplasm, of the syncytium in my first specimen is a little difficult to explain (Fig. 30). Exactly similar appearances are figured by Marchand, and by Aschoff and Apfelstedt, and described by Eden. In these cases they

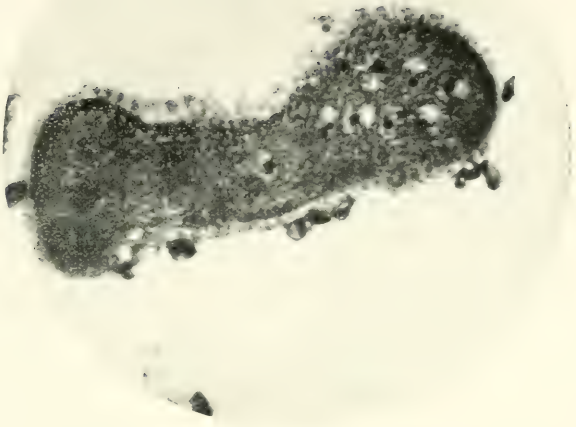

Fig. 28.—Striated border of syncytium. On the lower edge several adherent blood corpuscles may be seen.

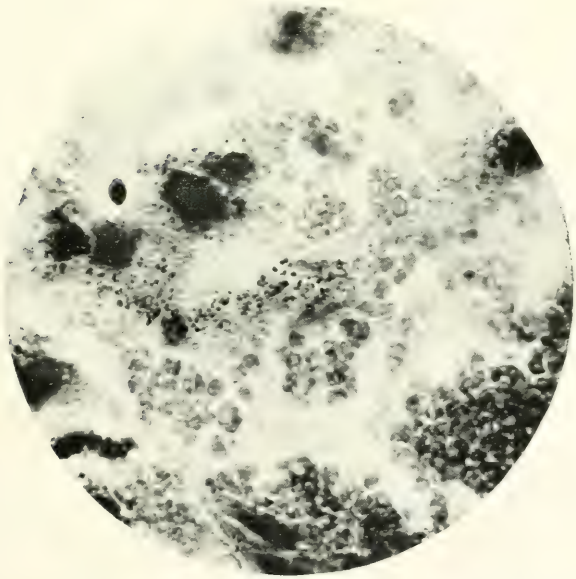

Fig. 29.—Syncytial strand surrounded by maternal blood. The foam-like structure of the syncytium may be seen, also the dark granules scattered through it. The outline of the syncytial strand is lost, as the lens was focussed on the granules.

[*Between pp.* 104-5.

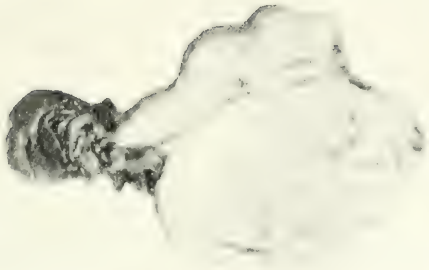

Fig. 30.—Ovary and tube with infundibular pregnancy.

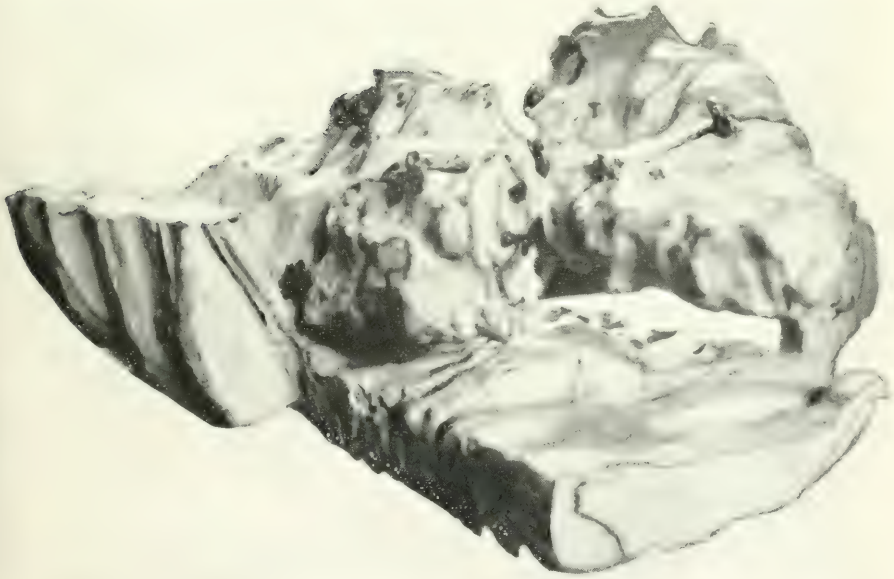

Fig. 31.—Wax reconstruction model (1-200) of the embryonic vesicles and their attachment to the blastocyst wall. The vesicles do not touch at any point.

are undoubtedly fat granules, the preparations being fixed in Müller's or Flemming's solutions and stained with osmic acid. My specimen was fixed with formalin, and passed through alcohol and xylol into paraffin, so that it is unlikely that any fat remained. Moreover, the granules appear in all the sections stained with hæmatoxylin and eosin. One slide I re-stained with osmic acid, but the granules remained exactly as before, and did not take on the osmic acid. It may be concluded, therefore, that they are not fat, however closely they resemble it in appearance and distribution. For glycogen none of my slides were stained, and that also may be put out of account.

Peters describes the presence of red and white blood corpuscles, both normal and degenerated, in the syncytium. But in his case this was an occasional occurrence, although it led him to the rather extreme conclusion that the blood cells were capable of taking part in the formation of syncytium out of their own elements. The appearances in my specimen are, however, quite apart from this. The granules are universally present throughout the syncytium and symplasma, and in practically no other tissues. Here and there I have seen isolated granules in the decidua or trophoderm cells. In the syncytium, on the other hand, they are so constant as to form in this particular specimen a fairly accurate means of recognising that tissue.

This uniformity and peculiarity of distribution make it quite clear that it is not an accidental deposit of pigment, and unlikely that it is a degeneration product. The close similarity of their arrangement to the fat figured by Marchand and Aschoff, and their translucent refractile appearance, led me to think that they might be lipoids in a crystalline state. Professor Lorrain Smith very kindly examined several sections with me, and by means of the polarising microscope demonstrated that this idea was wrong. He, however, suggested

another explanation which is very probably correct. In his view these granules, and the very similar granules seen in the spleen, are composed of molecules of blood-pigment adherent to a minute droplet of fat or lipoid. If the tissue is fixed and stained for fat, the granules appear as fat droplets. But in a specimen fixed like mine the lipoid nucleus is dissolved out, and the molecules of pigment are left adhering to one another and forming the granules that take on the eosin. This view I have not been able to test experimentally, but it is certainly the most likely explanation of this interesting appearance. It receives support also from the strong similarity of the granules to those described by Jenkinson as occurring in the trophoblast of the sheep. These he proved to consist of hæmoglobin derivatives from the ingestion and fragmentation of red blood cells. Bonnet regarded the pigment as hæmatoidin, but Jenkinson failed to confirm that opinion by means of the spectroscope.

This absorption of blood-pigment may, according to Lochhead be due either to the ingestion of red cells by the trophoblast, or to laking of the blood on the surface and the absorption of the hæmoglobin. He is unable to suggest the exact significance of the appearance of the granules. "Iron-free pigment is probably a waste product, while iron-containing pigment is stored in the fœtal organs. Whether the fœtus subsequently synthesises part of the organic iron compound into hæmoglobin, or absorbs minute quantities of hæmoglobin as such, according to its requirements, is unknown." At this very early stage the iron requirements of the embryo are probably slight, and the pigment may therefore be in process of being stored in the chorionic villi against later needs.

If it is true that these granules originally had a fatty nucleus, it indicates that from a very early stage the syncytium is able to take up fat for the benefit of the embryo. That it does so in the formed placenta is, of course, a recognised fact.

It is interesting to note that Eden mentions that he found fat in very occasional areas in the decidua and in the cytotrophoderm. This exact conformity with the distribution of the granules in my specimen is another argument in favour of the truth of Professor Lorrain Smith's view.

The cytotrophoderm is very similar in both specimens. The enlargement of the cells when they escape from the tips of the villi and spread out into the cell columns is most noticeable in the second specimen. It has also been recognised in several other ova.

The most interesting feature of the cytotrophoderm is the existence in the cell columns of empty spaces, presumably formed by the vacuolation of several adjacent cells (Fig. 11). This is clearly visible in the first ovum, and it is possible to demonstrate that they do not communicate with the intervillous space. In the second ovum there are numerous smaller spaces entirely devoid of contents, which exactly resemble other spaces lying quite near but full of blood. The presence of these larger spaces in the one ovum, and the coexistence of spaces in the second, some containing blood, others empty, seem to argue strongly in favour of the view that the ramifications of the intervillous space are formed by the opening out of such spaces in the trophoderm. This view receives strong support from the conditions found in the Bryce-Teacher ovum, and these observers believe that the spaces are first formed by the accumulation in the cell columns of the trophodermic secretion. This ultimately finds its way to the surface of the trophoderm and in so doing opens out the spaces, into which the blood then penetrates.

Another very strong argument as to the truth of this view is to be found in the fact that exactly similar spaces may be seen in the trophoderm in the rat and mouse—some, as in my specimen, quite empty, others filled with maternal blood; and the only conceivable way in which the maternal blood could

get into the spaces is by their opening out to the surface. This was pointed out by Professor Robinson some years ago, and I have actually seen several of his specimens which demonstrate the point.

The older view of Peters, that the spaces are formed by the outrush of maternal blood under pressure from the decidual vessels, after these had been opened into by the trophodermic activity, does not meet the observed conditions so satisfactorily.

The Embryonic Anlagen of the Intra-uterine Ovum.—It is most unfortunate that the rupture of the blastocyst and the consequent dislocation of its contents make it impossible to be certain as to the exact nature or arrangement of the two vesicles in this ovum. Before discussing the possible interpretations it may be well to enunciate the particular points that must be borne in mind in forming an opinion.

(1) (*a*) The first vesicle measures $0{\cdot}35 \times 0{\cdot}39 \times 0{\cdot}21$ mm.

(*b*) It consists of two layers, and at one point the outer layer is definitely continuous with the mesoderm of the blastocyst wall. The vesicle is thus quite clearly attached to the blastocyst wall.

(*c*) On one side of this rather triangular vesicle there is a distinct thickening, the cells being present in three or more irregular rows. The appearance at once suggests to the eye an embryonic area. The dimensions of this thickened area are $0{\cdot}21 \times 0{\cdot}2$ mm.

(2) (*a*) The second vesicle measures $0{\cdot}26 \times 0{\cdot}28 \times 0{\cdot}14$ mm.

(*b*) In structure it is a replica of the first vesicle.

(*c*) It is substantially attached to the blastocyst wall by the continuity of its outer layer with the blastocyst mesoderm at almost the farthest possible point from the first vesicle.

(*d*) At a level of about one-third of the way through this vesicle the cavity is almost completely filled up with cells, which form a floor, so to speak, dividing it into two parts.

(3) The attachments of the respective vesicles to the blastocyst wall are towards opposite ends of one side of that structure.

(4) The embryonic-area-like thickening is on the side of the first vesicle farthest removed from the second vesicle.

(5) Nowhere are the two vesicles in contact. Where they are most approximated to each other there is no trace of loose cells which might be regarded as the broken remains of an embryonic area between the vesicles.

Naturally the first endeavour is to translate the contents of the blastocyst as parts of a single normal embryo. In doing so several difficulties are met with. In the first place, the two vesicles are entirely detached; and secondly, there is no indication of any cells that might originally have formed a bond between them. Thirdly, both vesicles are attached to the blastocyst mesoderm at points considerably separated. Fourthly, both vesicles are identical in structure, and there is no suggestion in their structure as to which might be amnion and which yolk sac. Fifthly, while the whole ovum corresponds very closely in its general development and in the dimensions of the blastocyst to that of Jung, yet each individual vesicle is about the same size as the whole embryonic anlagen (amnion and yolk sac combined) in Jung's specimen. Jung's exact figure is 0·25 mm. measured by the number of sections through which the embryonic structures stretched. Later he points out that the yolk sac only stretches through twelve sections, each about 13 micromillimetres thick, giving for that vesicle a length of about 0·15 mm. The exact length of the two vesicles in the present ovum,

as measured by the number of sections of known thickness, is 0·35 and 0·26 mm. respectively.

In regard to the first three difficulties it may be argued that the same applies to the Bryce-Teacher ovum. There the two vesicles are even more widely separated, without any trace of connection between them, and each is attached to the mesoderm. So far as it goes that is perfectly true, but that ovum was so young that one would hardly expect any very definitely formed embryonic area intervening between the two sacs, and the attachments to the mesoderm are of the most slender variety. Further, the Bryce-Teacher ovum represents a stage before the splitting of the mesenchyme to form the extra-embryonic cœlom, and therefore the attachments may be partly accidental, due to the shrinkage and condensation of the mesenchyme in the hardening. The present ovum, on the other hand, represents a stage after the formation of the extra-embryonic cœlom, and in spite of that both vesicles have definite attachments to the mesoderm.

In regard to the fourth difficulty, it must be remembered that in Peters' and Jung's ova there is a definite differentiation of the cells at the base of the amnion, indicative of the formation of the embryonic ectoderm. The only situation in which there is anything similar in my ovum is the thickening on the wall of the first vesicle at the side most remote from the second sac.

One other possible view in regard to the attachment of both vesicles is that one attachment represents an amniotic duct. This is a duct or cord of cells connecting the amniotic cavity with the outer surface of the chorion. Eternod, Marchand, and Beneke have described what they believe to be indications of it in human ova, and Selenka has described it in the ova of apes. According to Keibel, it is very doubtful if it is at all a regular condition in the human

ovum. Probably not, for it is but "a phylogenetic memory from dim ancestral times." In specimens such as Marchand's there is a depression on the surface of the chorion at the end of this duct. In the case of the second vesicle in my specimen there is no depression, and nothing to support such a view. In regard to the first vesicle I cannot speak so definitely, as the chorion has been ruptured in the immediate neighbourhood of the attachment of the sac. But so far as I can see, there is no suggestion of a depression. In any case, even if the first sac was clearly proved to be amnion, there still remain the other difficulties in the way of co-ordinating the two sacs as parts of one single embryo.

Failure to interpret the vesicles satisfactorily as parts of a single embryo compelled one to study them as the possible components of a twin ovum. The interpretation involves three assumptions. The first is, that the thickening on the wall of the first vesicle is an embryonic area. The second is, that on the other side of it there was another sac—probably the yolk sac—which has been broken up and dispersed. In Fig. 18 there is shown a strand of cells stretching out towards the second vesicle, which, if curled round the opposite way, might be imagined to have formed a closed sac superficial to the thickened area.

The third assumption is, that the cells filling up the cavity of the second vesicle through the thickness of several sections do really form a floor, and represent the embryonic area of the second embryo. In this event the area must have been cut on the flat, and that part of the vesicle superficial to it would represent the yolk sac, while the part beneath the embryonic area would represent the amnion. This interpretation would bring the second embryo into very close correspondence with the dimensions of Jung's embryo.

In connection with this view the investigation of the specimen gave a useful example of the value of studying such

a specimen, not only by serial sections, but also when possible by means of a model reconstruction (Fig. 31). For the construction of a wax model on a scale of 1:200 of the two vesicles and the side of the blastocyst to which they are attached, I am greatly indebted to Professor Robinson, who personally made nearly two hundred drawings with the camera lucida, and reconstructed the model. One of the special points that the model cleared up was in connection with a fold or pocket of the first vesicle at the base of the thickened area. The study of some isolated sections, in which this fold appears as a separate vesicle on the surface of the thickened area, would have led us to regard the fold unhesitatingly as the yolk sac of a first embryo, and would have rendered the interpretation of the specimen as one of uniovular twins perfectly clear and free from any objection. By studying serial sections, however, and still more by the construction of a model in three dimensions, it became obvious that this pocket or fold was directly continuous with the rest of the vesicle. We were thus led to avoid a somewhat tempting error! In other respects the model fully confirmed our previously formed views as to the relations and shapes of the vesicles, and the fact that they do not touch at any point.

The conclusion of the whole question is, I believe, that it is impossible to determine with any approach to certainty in favour of either the one interpretation or the other. The specimen may be one of a single embryo, with or without an amniotic duct, whose component parts have been seriously dislocated by mechanical influences, probably during the removal of the uterus from the body. On the other hand, it may be a specimen of uniovular twins at a very early stage of development, the yolk sac (?) of the first embryo being broken up and dispersed. The arguments on either side seem to be fairly well balanced. The decision of any individual observer must remain merely an opinion.

The Age of the two Ova.—In neither case have I been able to get any history that would help in determining the age of the ovum. In the first case the husband did not know the date of the last period, although he was quite sure that no period had been missed. Before I could interrogate him more closely he had left the city. In the case of the second specimen, the patient had forgotten the exact dates by the time I came to investigate the question in detail. All she knew was that the last period had been "about three weeks before the operation."

Any estimation of the age must therefore be made by the rather tentative method of comparison with other specimens. The first specimen corresponds closely to Jung's in its dimensions, and in the development of the villi. That ovum, according to the table of Bryce and Teacher, whose reckoning I regard as more accurate than that of His in spite of such cases as Giacomini's "eleven days'" ovum, was probably about fourteen and a half to fifteen and a half days old. I would therefore place the age of my first specimen at about fifteen days.

I have figured and described the corpus luteum, for, as Mall points out, no record of an ovum is complete without it, if it is available. In size and development it corresponds fairly closely to the description of the corpus luteum in the second week given by Leopold and Ravano. So far as it goes, therefore, the corpus luteum supports this estimate of the age of the ovum, but I do not think that we are as yet in possession of anything like an accurate standard of the development of that strange organ, and I do not place much importance upon it.

The blastocyst of the second specimen measures $2{\cdot}73 \times 6 \times 5{\cdot}6$ mm. The embryonic area proper measures 1·6 mm. The blastocyst therefore corresponds to the specimens of Rossi Doria and Eternod, which Bryce and Teacher place at eighteen to nineteen days old. The embryo

in regard to development lies between "Gle" of von Spee and "Klb" of Kroemer and Pfannenstiel. The former is regarded by Bryce and Teacher as nineteen to twenty days old. I think therefore that the second ovum was probably about twenty days old.

In conclusion, it is a great pleasure to express my thanks to Professor Harvey Littlejohn and Dr William Fordyce, from whom the respective specimens were obtained, and to Dr Barbour for permission to investigate the first ovum. Professor Arthur Robinson has allowed me to draw freely on his experience and knowledge, and, as stated, personally gave much time to the reconstruction of the model. For all this help, so willingly given, I offer him my heartiest thanks. I am indebted to the Earl of Moray Endowment of the University for a contribution to the expenses of the research, and to the Carnegie Trust for part of the cost of the illustrations.

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The Vice-President said they were very much indebted to Dr Johnstone for his interesting communication. Comparatively rarely had one the opportunity of investigating an early ovum *in situ*. In this case the patient died somewhat suddenly from a heart condition, and, by a lucky chance, the uterus was opened and the specimen discovered. Dr Johnstone was also to be congratulated on his demonstration; personally he had looked

at many text-books and seen many demonstrations, but had never seen such a beautiful one as the epidiascope had revealed. The case was successfully established as one of early pregnancy, whether a possible twin pregnancy, as Dr Johnstone said, one could not dogmatise.

Dr Berry Hart said Dr Johnstone had given a very clear account of an exceedingly interesting case. The microscopical specimens were very good, especially those relating to the development of the placenta. He agreed that one could not settle the point as to whether or not there was a twin. As to the site of the embryo, the part which Dr Johnstone indicated looked very like that given in Peters' ovum, where there were two layers of cells. One peculiar thing was that in Dr Johnstone's specimen one would have expected to have seen the amniotic cavity lying above the double layer of cells. His (Dr Berry Hart's) impression was that it was an undoubted pregnancy and possibly twin, but he thought the embryonic structures must have died some time before the woman. They had in these specimens really a very good representation of the origin of the placenta, but the real embryonic part had degenerated. The points brought out with regard to the villi confirmed what they already knew. In connection with the maternal blood supply, he understood Dr Johnstone to say that in some sections there was an endothelial lining. That was the old view, but the whole demonstration seemed to favour the new view. He regretted that the specimen was not better; had Dr Johnstone got a perfect specimen, from the ability with which he had dealt with this one, it would undoubtedly have been very valuable. He suggested that Dr Johnstone should compare the structure of the villi and intervillous spaces with those of Goldmann's *intra vitam* injections in mice.

Dr Haig Ferguson had only to thank Dr Johnstone very much for his interesting illustrations, which he had enjoyed

exceedingly. He had almost proved that there was early twin pregnancy. The trouble he had taken and the excellent specimens produced were worthy of all praise.

Dr James Ritchie also thanked Dr Johnstone for his paper, and agreed with Dr Berry Hart that if he had had a perfect specimen the demonstration would have been still better worth listening to; he also associated himself with the Vice-President in saying he had never seen such a fine demonstration of early pregnancy.

Dr W. Fordyce joined with the previous speakers in his appreciation of Dr Johnstone's demonstration.

Dr Young, in view of a somewhat similar case which he brought before the Society some years ago, said, firstly, he wished to congratulate Dr Johnstone on his great success, and deplored with him the rather unfortunate way in which the specimen had been presented for investigation. Personally, he could not recollect having seen a more beautiful demonstration before.

In regard to the anatomical points, the specimen in many ways, as Dr Johnstone indicated, very closely counterfeited the one which Jung so fully described some years ago. As some of the members would recollect, a year or two ago, he (Dr Young) had the opportunity of showing a very early embryo—one at an earlier stage of pregnancy than this one. The earliest ovum was described some years ago by Bryce and Teacher, and his (Dr Young's) case was an intermediate link between Bryce and Teacher's and Peters', *i.e.*, it was the second earliest ovum ever described. In his case the mesoderm was scattered throughout the entire extent of the blastocyst, and the decidual reaction was extremely meagre.

The changes which he had then fully described and which Dr Johnstone referred to and so clearly demonstrated, had

amply convinced him that one had to look to a different explanation of the method of opening out of the maternal vessels and the establishment of the blood supply than that set forth in the orthodox text-books. As he (Dr Young) had demonstrated, it was an extremely simple thing to see that as these distended capillaries gradually extend they inevitably must result in pouring their contents into the implantation cavity. He had suggested this some years ago, and he believed that this view was now accepted. Dr Johnstone's specimen had again clearly demonstrated these facts.

It was very fortunate that the cellular preservation was so good in this specimen; in his own case it was not so good, and it was extremely surprising that it should be so good in view of the time which intervened between the death of the patient and the *post-mortem*. Had Dr Johnstone carried his investigations into the endometrial changes; and, if so, had he noticed the vacuolation change? He (Dr Young) did not think it would be possible to detect it in this specimen, for it was only present at the earliest stages. The decidual reaction soon obscured it.

Dr Johnstone, in reply, said he was very much indebted to the Fellows of the Society for the extremely kind way they had spoken. With Dr Berry Hart's view that perhaps the embryo had died before the woman, he was not inclined to agree, because the embryonic tissue was in better preservation than any of the outer portion of the ovum. Although this ovum might, according to the strictest canons, be classified as pathological, personally he thought the pathology was due to mechanical violence during removal of the uterus.

With regard to Dr Young's question, he had not to any great extent examined the endometrium with that special point in view. There was no congestion, but œdema throughout. He would be most happy if Dr Young would come and see the specimen and examine it for himself on that point.

VI. BIOMETRIC ANALYSIS OF SOME INSEMINATION-LABOUR AND MENSTRUAL-LABOUR CURVES IN CERTAIN MAMMALIA.

By D. Berry Hart, M.D., F.R.C.P.E., Lecturer on Midwifery and Diseases of Women, Surgeons' Hall, Edinburgh.

In a previous communication[1] I showed that from Tessier's statistics as to the insemination-labour duration in ewes, from Earl Spencer's in cattle, and from von Winckel's and Reid's menstrual-labour durations in women, a frequency polygon could in each case be constructed.

In Tessier's ewes the curve from 912 labours was a lofty symmetrical one, slightly skew on the right side. The dates were given in 24-hour intervals, and were continuous over 11 days.

In the others (Spencer's, von Winckel's, and Reid's) the curve was interrupted by peaks when grouped in 24-hour intervals, but was still of a frequency nature. When Spencer's were taken in 48-hour groupings, the curve smoothed out (Fig. 1). In von Winckel's and Reid's a 96-hour grouping gave the same result, *i.e*, a fairly smooth and symmetrical frequency curve was obtained (Fig. 3 gives Reid's).

The irregularities in the Spencer, von Winckel, and Reid statistics seemed to me to be due to the close 24-hour grouping and to the consequent separation of births happening near midnight from those quite close to them shortly after midnight. By grouping them in 48- or 96-hour periods this mal-allotment may have been avoided to a great extent.

[1] "On the Duration of the Interval between Insemination and Parturition in Certain Mammals as Studied by Biometric Curves, with Special Reference to the Calculation of the Onset of Labour in Human Pregnancy." *Edin. Obstet. Trans.*, xxxviii., 107; *Edin. Med. Journ.*, 1913, xi., 291.

The question still arose, however, as to the propriety of smoothing out these curves by a means of which the method was not quite clear, and therefore one had to face this point—Given 24-hour groupings with the peaked irregularities, what could be made of them by biometric treatment—would Gauss's method of least squares be of use?

I therefore consulted my friends Mr J. D. Hamilton Dickson, Fellow and Tutor of Peterhouse, Cambridge, and Dr A. Daniell, whose *Text-Book of Physics* is well known, as to this, and was fortunate enough to enlist their sympathy and active help in the question. Mr Dickson worked out Spencer's and Reid's data, and Dr Daniell treated the question from the curve point of view, so that from their most valuable help I am now enabled to bring the matter up again with benefit to the elucidation of the practical obstetrical questions —What is the significance and accuracy of a labour date calculated from a single insemination in cows, or from the last day of the last menstrual period in the human female? What is the most probable insemination-labour duration in cattle and the most probable menstrual labour duration in the human female? These data may be regarded as the varying measurements between insemination and labour or between the last menstruation and labour, and can therefore be treated by Gauss's method of least squares, a form of mathematical inquiry used in astronomical observation data, and, in general, by statistical observers, to settle, in any series of observations of the same or similar objects, the most probable result, *i.e.*, the arithmetical mean of the observations, its probable error as well as the probable error of a single observation. I do not propose to explain this method in all its bearings, as that would involve points unnecessary here, and is best studied in such works as Merriman's *Method of Least Squares*, Jevon's *Principles of Science*, and Herschell's article in the *Edinburgh Review* for 1850.

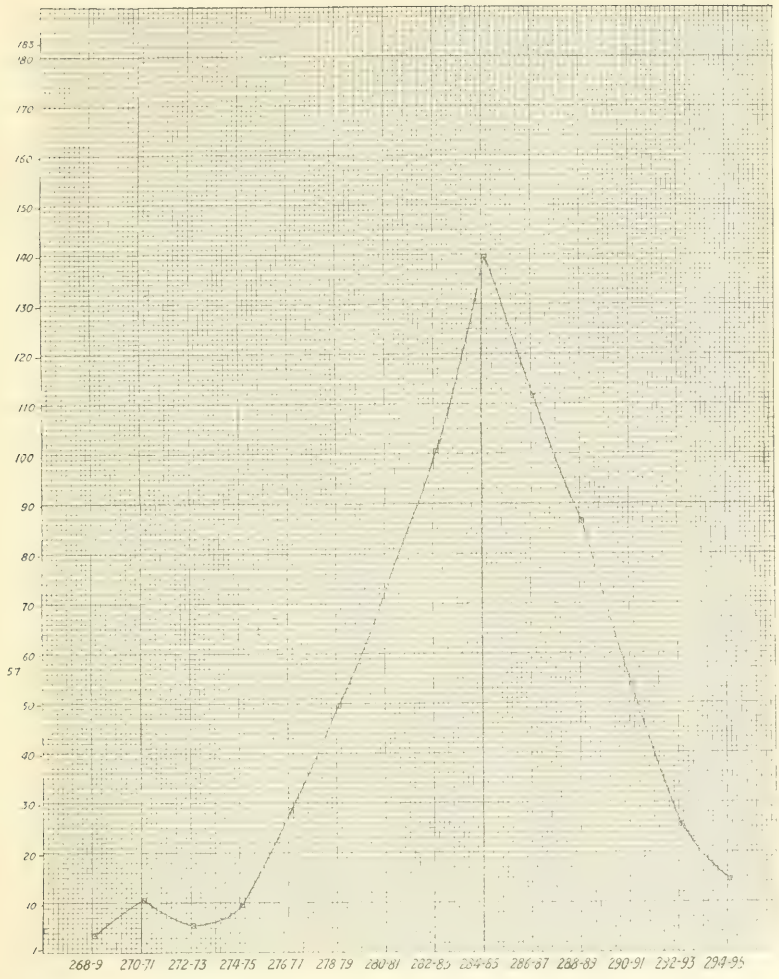

FIG. 1.—Frequency polygon based on Earl Spencer's birth statistics of 764 cattle, in 48-hour groupings.

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[Between pp. 120-21.

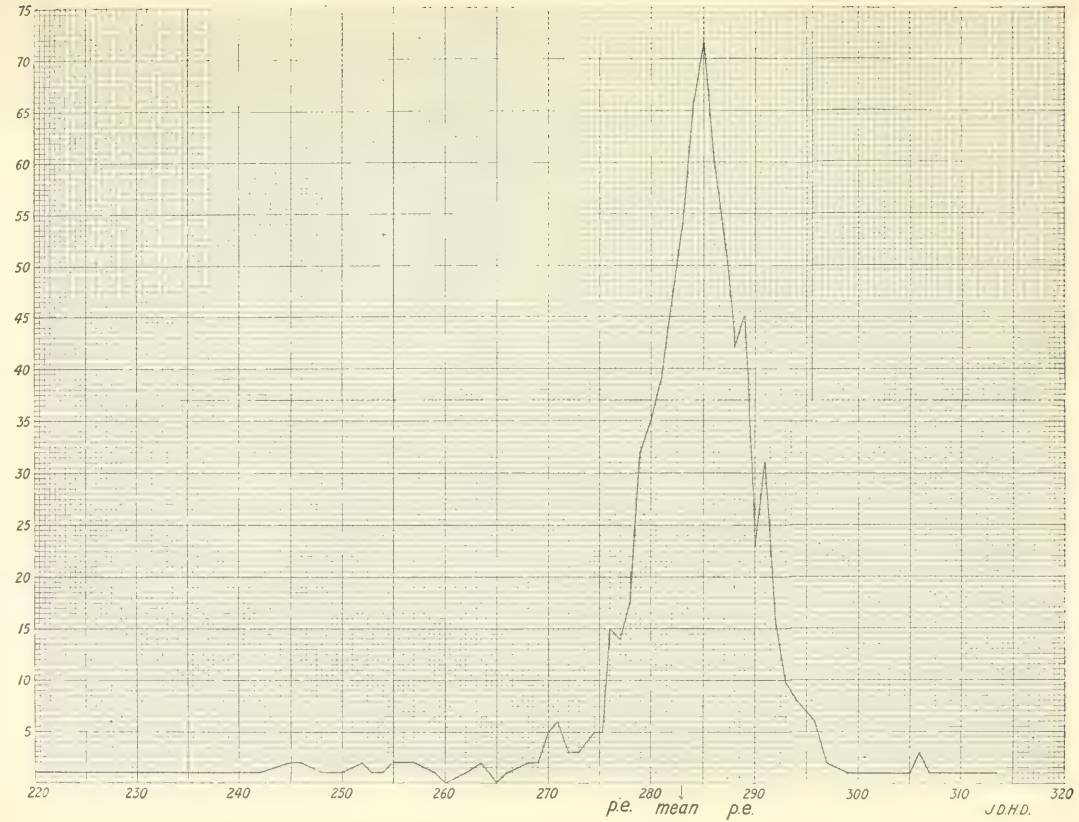


FIG. 2.—Spencer's cattle statistics in 24-hourly observations.

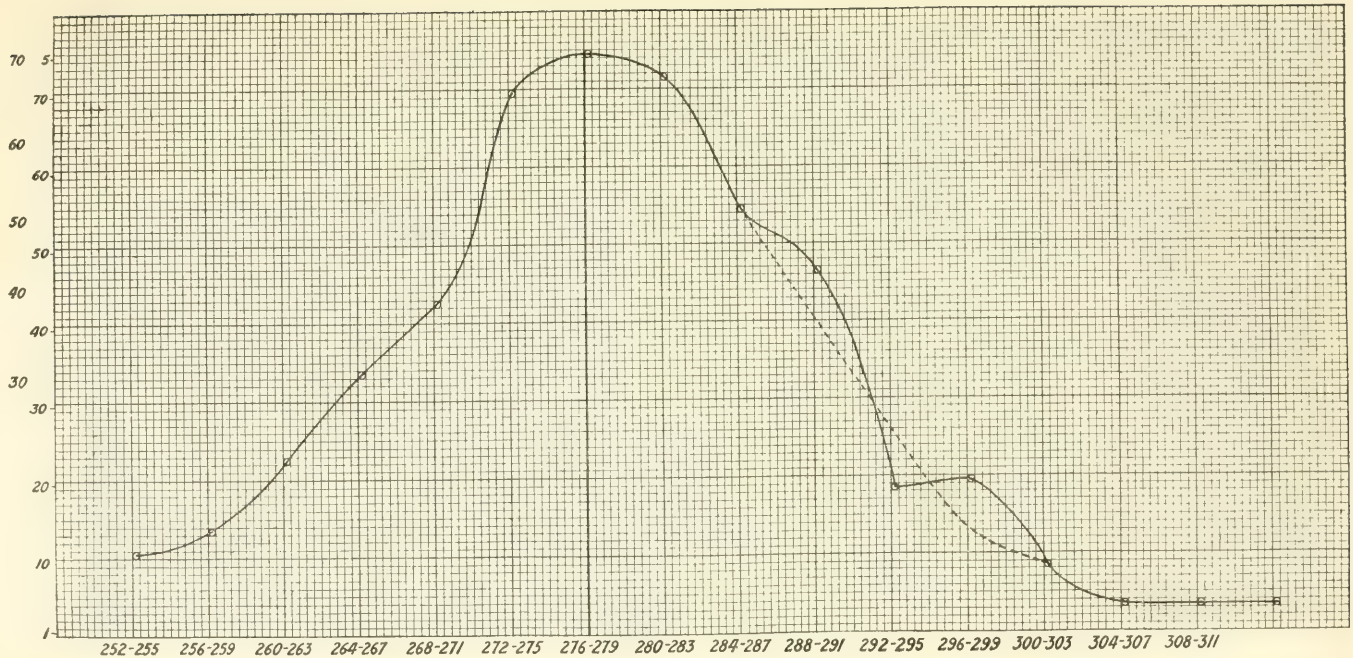


Fig. 3.—Frequency curve based on Reid's statistics of 500 human female births, in 96-hour groupings, and calculated from last day of last menstruation.

Jevons gives the following summary of Gauss's method, and I have added a few explanatory remarks:—

1. Draw the mean of all the observed results (by dividing the sum of the measurements by their number).

2. Find the excess or defect, that is, the error of each result from the mean (call this v).

3. Square each of these results (and call each v^2 and the sum of them Σ).

4. Add together all these squares of the errors, which, of course, are all positive.

5. Divide by one less than the number of observations. This gives the *square of the mean error.*

6. Take the square root of the last result; it is the *mean error of a single observation.*

7. Divide now by the square root of the number of observations, and we get the *mean error of the mean result.*

8. Lastly, multiply by the natural constant 0·6745 (or approximately by 0·674, or even by $\frac{2}{3}$), and we arrive at the *probable error of the mean result.*

I now go on to give Mr Hamilton Dickson's calculations in Spencer's cases of 764 cattle, and in Reid's 500 human menstrual-labour durations in 24-hour intervals. Mr Dickson also plotted out these results on graph paper, and drew the frequency polygon threading Reid's (Fig. 4).

If we deal first with the numerical results of Spencer's cattle statistics, it is to be noted in Mr Dickson's analysis below that in the first column (left hand) the separate number of days is given in each case; in the second the number of cattle which had the same duration; in the third the product of the number of cattle and the number of days these cattle gestated, and at the foot the sum,

216,451, or the Σ in such an expression as $\sqrt{\frac{\Sigma(v^2)}{n-1}}$;

in the fourth the difference between the mean and each

result (v); in the fifth the squares of the differences (v^2); and in the sixth these multiplied by the number of cases. The sum of the squares of the differences is 60,629. I now go on to quote Mr Hamilton Dickson's text, and his interesting comment.

The probable error of *one* observation is (r),

$$0{\cdot}6745\sqrt{\frac{\Sigma(v^2)}{n-1}}$$

where n is the number of observations, and v is as defined above. This arithmetical mean is 283·3; but to simplify the calculation (we shall see that it does not affect the result later) it was taken as 283.

Hence
$$\Sigma(v^2) = 60{,}649.$$
$$\frac{\Sigma(v^2)}{763} = 79{\cdot}48.$$
$$\sqrt{\frac{\Sigma(v^2)}{763}} = 8{\cdot}915.$$

and then
$$r = 0{\cdot}6745 \times 8{\cdot}915 = 6{\cdot}012.$$

This quantity r is mainly useful for drawing the probability curve; but as your purpose is the period of gestation, and the accuracy to be obtained from these observations, we are more concerned with the probable error of the *mean* of the observation, viz. 283·3; this *p.e.* is (r_0), given by

$$\begin{aligned} r_0 &= 0{\cdot}6745\sqrt{\frac{\Sigma(v^2)}{n(n-1)}} \\ &= 0{\cdot}6745 \times \sqrt{0{\cdot}1041} \\ &= 0{\cdot}6745 \times 0{\cdot}323 = 0{\cdot}218. \end{aligned}$$

The result is that, taking all the observations, their mean is 283·3 (or 283) days, with a probable error amounting to 0·2 of a day, and therefore we may say 283 days net.

FIG. 4.—Reid's statistics of 500 cases in 24-hour dates, with symmetrical frequency curve threaded through them.

[*Face page* 122.

Spencer's Observations Calculated from Insemination.

			σ	σ^2	
220	1	220	63	3,969	3,969
..					
226	1	226	57	3,249	3,269*
..					
233	1	233	50	2,500	2,500
234	1	234	49	2,401	2,401
235	1	235	48	2,304	2,304
..					
239	1	239	44	1,936	1,936
..					
242	1	242	41	1,681	1,681
..					
245	2	490	38	1,444	2,888
246	2	492	37	1,369	2,738
..					
248	1	248	35	1,225	1,225
..					
250	1	250	33	1,089	1,089
..					
252	2	504	31	961	1,922
253	1	253	30	900	900
254	1	254	29	841	841
255	2	510	28	784	1,568
..					
257	2	514	26	676	1,352
258	3	774	25	625	1,875
259	1	259	24	576	576
..					
262	1	262	21	441	441
263	2	526	20	400	800
..					
266	1	266	17	289	289
..					
268	2	536	15	225	450
269	2	538	14	196	382
270	5	1,350	13	169	845
271	6	1,626	12	144	864
272	3	816	11	121	363
273	3	819	10	100	300
274	5	1,370	9	81	405
275	5	1,375	8	64	320
276	15	4,140	7	49	735
277	14	3,878	6	36	504
278	18	5,004	5	25	450
279	32	8,928	4	16	512
280	35	9,800	3	9	315
281	39	10,959	2	4	156
282	47	13,254	1	1	47
283	54	15,282	0	0	0
284	66	18,744	1	1	66
285	74	21,090	2	4	296
286	60	17,160	3	9	540
287	52	14,924	4	16	822
288	42	12,096	5	25	1,050
289	45	13,005	6	36	1,620
290	23	6,670	7	49	1,127
291	31	9,021	8	64	1,984
292	16	4,672	9	81	1,296
293	10	2,930	10	100	1,000
294	8	2,352	11	121	968
295	7	2,065	12	144	1,008
296	6	1,776	13	169	1,014
297	2	594	14	196	392
..					
299	1	299	16	256	256
..					
304	1	304	21	441	441
305	1	305	22	484	484
306	3	918	23	529	1,587
307	1	307	24	576	576
..					
313	1	313	30	900	00
	764	216,451			60,649

Mean = 283·3

* This is a *lapsus pennæ* for 3249, and it has been left, as it involves only $\frac{1}{3000}$ error.

On the curve it *looks* as if 285 is nearer it; but there is no good reason for rejecting the twenty-five observations from the 220th to the 259th day inclusive. *If*, however, we reject them, then the mean day is 285·8—say the 286th—which errs again by being (from the curve) apparently a little too high.

For my own part, I do not value 286 as so good a result as 283; but the physical reasons may perhaps justify the preference for 286. I have not calculated the *p.e.* for 286, but, in any case, it can only be a small part of a day.

The smallness of r (only ± 6 days, on 283) indicates that the observations give a very close approximation to the truth; it means that half of the births fell between the 276th and 289th days. The curve would be a very *thin stand-up* one, but I have not calculated it.

The probable error (r) of one observation is

$$r = 0{\cdot}6745\sqrt{\frac{\Sigma(v^2)}{n-1}}$$

Here $\Sigma\ (v^2) = 65{,}829$, with the *mean* 278 (more accurately 278·276), $n = 500$.

$$\therefore \quad \sqrt{\frac{\Sigma(v^2)}{n-1}} = \sqrt{\frac{65{,}829}{499}} = \sqrt{131{\cdot}922} = 11{\cdot}49$$

hence $$r = 0{\cdot}6745 \times 11{\cdot}49 = 7{\cdot}748.$$

Also probable (r_0) of the mean is

$$r_0 = 0{\cdot}6745\sqrt{\frac{\Sigma(v^2)}{n(n-1)}}$$
$$= 0{\cdot}6745 \times 0{\cdot}514 = 0{\cdot}3466.$$

The result is—the most probable menstrual-labour duration of gestation is 278 days (or, if you like, 278·276 days), with a probable range of 0·35 (0·3466) of a day, more or less. In other words, the most probable duration lies between

278·28 − 0·35 and 278·28 + 0·35.
277·93 days and 278·63 days.

Reid's Durations in 500 Labours Calculated from the Last Day of Menstruation.

			v.	v^2.	
252	4	1,008	26	676	2,704
253	1	253	25	625	625
254	3	762	24	576	1,728
255	1	255	23	529	529
256	2	512	22	484	968
257	4	1,028	21	441	1,764
258	4	1,032	20	400	1,600
259	4	1,036	19	361	1,444
260	6	1,560	18	324	1,944
261	5	1,305	17	289	1,445
262	3	786	16	256	768
263	9	2,367	15	225	2,025
264	10	2,640	14	196	1,960
265	5	1,325	13	169	845
266	10	2,660	12	144	1,440
267	9	2,403	11	121	1,089
268	13	3,484	10	100	1,300
269	5	1,345	9	81	405
270	13	3,510	8	64	832
271	12	3,252	7	49	588
272	13	3,536	6	36	468
273	16	4,368	5	25	400
274	21	5,754	4	16	336
275	20	5,500	3	9	180
276	16	4,416	2	4	64
277	16	4,432	1	1	16
278	22	6,116	..	.	..
279	21	5,859	1	1	21
280	15	4,200	2	4	60
281	18	5,058	3	9	162
282	25	7,050	4	16	400
283	14	3,682	5	25	350
284	15	4,260	6	36	540
285	14	3 990	7	49	686
286	15	4,290	8	64	960
287	11	3,157	9	81	891
288	17	4,896	10	100	1,700
289	8	2,312	11	121	965
290	9	2,610	12	144	1,296
291	14	4,074	13	169	2,366
292	6	1,752	14	196	1,176
293	3	879	15	225	675
294	6	1,761	16	256	1,536
295	2	590	17	289	578
296	5	1,480	18	324	1,620
297	8	2,376	19	361	2,888
298	6	1,788	20	400	2,400
299	1	299	21	441	441
300	2	600	22	484	968
301	4	1,204	23	529	2,116
302	1	302	24	576	576
303	1	303	25	625	625
304	1	304	26	676	676
305	2	610	27	729	1,458
306	0	0	28	784	0
307	1	307	29	841	841
308	2	616	30	900	1,800
309	0	0	31	961	0
310	1	310	32	1,024	1,024
311	1	311	33	1,089	1,089
..					
314	1	314	36	1,296	1,296
315	2	630	37	1,369	2,738
316	1	316	38	1,444	1,444
	500	139,138			65,829

Mean = 278·3

The following is Mr Dickson's comment on the results:—

PETERHOUSE, CAMBRIDGE,
22nd November 1913.

DEAR DOCTOR BERRY HART,—I have calculated *Spencer* and *Reid* in each case for the *whole* number of observations given.

Spencer.—The most probable length of the insemination-labour period is 283·3 days, with a probable variation, up or down, of 0·22 of a day. If we use only the observations from the 262nd to the 313th day inclusive, then the most probable result is 285·8 days, with a somewhat similar variation as in the other case, but I have not calculated it, as the calculation would have to be made almost *de novo*, and it is long. Besides, cows are not so interesting as humans, and for a like reason I have not calculated the probability curve (Fig. 2).

Reid.—Here the most probable period of gestation is 278·276—say 278—days, with a probable variation, up or down, of 0·347 of a day. I have plotted the probability curve [1] in the midst of the observations, and you see it fits in very comfortably.

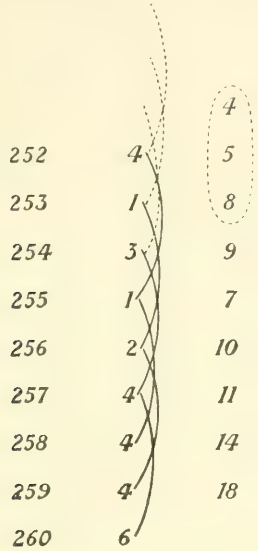
Of course the humans' observations cannot be so exactly got as those of cows, so that the cows' curve is much sharper than the humans'; that is to say, we can determine more accurately for cows than for humans—a result of which we have the measure in the two, probable errors, 0·22 and 0·347, almost in the ratio of 2:3 *i.e.*, cows are $1\frac{1}{2}$ times more accurate than humans.

With regard to "smoothing" observations, I am always against it, my view being that the observations treated (as here) by least squares will look after themselves better than any plan we can adopt. But if we want to save calculation and (as in the present case) reduce[2] the amount of numbers to be employed (say) by grouping in 48 hours or in 4 days, then

[1] Calculated from these observations.

[2] I plot all these 4-day points, then I use judgment in cutting out very astray ones, and calculate with the rest. But, after all, I prefer taking the actual observations themselves.

the plan is (for 4 days) to take *every* adjacent 4 days possible. Thus Reid, p. 406, I should treat as below.



and so on omitting

4, 5, 8.

The general theory of probability allows for no exceptional cases, *i.e.*, it expects to involve every case, however bad *we* may think it. But if we know of any reason attached to a particular case which makes it obviously unreliable, then, of course, cut it out. At the same time Chauvenet (an American mathematician) has devised a criterion by which a test can be applied for cutting out doubtful observations. However, it practically amounts almost to a recalculation for each observation cut out, and is too laborious to be used in actual observations.—Yours sincerely, J. D. HAMILTON DICKSON.

In the graph of Reid's statistics Mr Dickson has threaded a symmetrical frequency curve, and this shows clearly the

theoretical curve and the actual data plotted out in the peaked lines. Any point on the curve would satisfy the equation to the curve. Gauss's method of least squares gives us the power of settling the probable error in about half the cases, those grouped round M and between *p.e.* and *p.e.*

Spencer's 24-hour graph is seen at Fig. 2 and the smoothed-out one at Fig. 1.

I may now discuss some points relative to the value of the results given by the smoothed-out curve and the 24-hour unsmoothed results when treated by the method of least squares. On this Dr Daniell has given me valuable notes. In some respects they are too technical for actual reproduction, but I have greatly benefited by his discussion. Reid's smoothed-out data (Fig. 3) show that they form a frequency polygon, and that the most births occur between the 271st and 291st day. Mr Dickson's results are for the same data, and give a mean of 278 days, with a probable error of 7 days on each side. The most probable duration of the menstrual-labour period is 277·93 and 278·63 days—practically 278 days.

It will be noticed that in the smoothed-out frequency polygon of Reid's data there is a humped irregularity in the 288-291-day group, and also a balancing one in the 292-295 day group. This probably means some disturbance of the average position of the ovum at a late period after insemination. Thus Venn remarks in his *Logic of Chance* on height data in English, French, and Belgian adults, according to Quetelet's statistics, that the mean in Englishmen is 5 ft. 9 ins., in Belgians about 5 ft. 7 ins., and in Frenchmen about 5 ft. 4 ins. The mixture of all these data would give a disturbing hump on the right side owing to their heterogeneity. The same would happen if the wafer on the target were displaced a certain distance after a few thousand shots had been fired, *i.e.*, the curve derived from the whole data would be skew, as Venn figures. Thus the skew nature of Reid's polygon might be

explained by a late altered situation of the shed ovum, *e.g.*, its being in a deep fold of the mucosa.

It is evident, then, that one will find Gauss's method of greater value, but the plotting and smoothing out of curves has some advantages, and is a graphic appeal to the eye, of the generalisation, that the dates of labour after insemination or after a menstrual date conform to the law of probability within certain limits; that beyond these limits we get results of a less calculable nature, but all the same, results that can be understood on the principles just laid down.

I here append an obstetric calendar (p. 130) based on the results given above. It will be readily understood. Thus, if the last day of the last period was 6th January, 11th October, the date below the 6th, would be in the centre of the most probable fortnight for the occurrence of labour, that is, the most probable date would be seven days before or after the 278th day—in this case 11th October. It might, however, occur earlier or later than this fortnight, but this could not be foreseen, although understandable on the principles laid down.

Literature.

Galton, Sir Francis.—*Memories of My Life*, p. 302 (London: Methuen & Co.); also "Family Likeness in Eye Colour," *Proc. Roy. Soc. Lond.*, 1886, for Mr Dickson's appendix to Galton's paper.

Jevons, W. S.—*The Principles of Science*, p. 208 (Macmillan & Co., 1877).

Venn.—*Logic of Chance*, 3rd ed. (Macmillan & Co., 1888).

For additional literature, see author's paper, *Edin. Med. Journ.*, October 1913.

The Vice-President was sure they were all very much indebted to Dr Berry Hart for his obstetrical and at the same time deep mathematical paper. One noticed that in finishing

Obstetrical Table.

January	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
October	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	November
February	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28				
November	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	1	2	3				December
March	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
December	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	January
April	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30		
January	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2		February
May	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
February	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	1	2	3	4	5	March
June	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30		
March	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4		April
July	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
April	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	1	2	3	4	5	May
August	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
May	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	June
September	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30		
June	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	1	2	3	4	5		July
October	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
July	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	August
November	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30		
August	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4		September
December	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	
September	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	1	2	3	4	5	October

This table is based on a 278-day menstrual-labour duration for the human female. The initial date taken is the last day of a menstrual period.

up Dr Berry Hart allowed ten days or a fortnight's difference. They would be glad to hear what some of the other Fellows had to say, especially some of those who could appreciate the mathematical standard much more clearly than he could.

Dr Haig Ferguson had nothing to add. He had listened with great pleasure to the paper, although he could not say he had followed Dr Berry Hart. The only practical point was that he (Dr Haig Ferguson) generally calculated from the first day of the last menstrual period, and the duration of the period got over the necessary number of days. It was a very simple calculation, and perhaps more accurate than counting from the end.

Dr Lamond Lackie asked if in Dr Reid's statistics both multiparæ and primiparæ were included? In primipara the duration was sometimes shorter.

Dr Berry Hart replied that there was no distinction at all.

Dr Lamond Lackie asked if he believed in it.

Dr Berry Hart replied that he did not in the slightest.

Dr R. W. Johnstone said Professor Robinson, who had been investigating the case of ferrets, had told him he had found that if one took a number of ferrets that were inseminated on the same day and then killed them at an early stage of development, the embryos were found to be in considerably different stages of development. But when a similar number of ferrets were allowed to go on till full time, then the insemination labour curve was practically uniform, all the animals going into labour on practically the same day. Apparently from that statement development must go on at different rates in different embryos, and there must in some instances be a "speeding up" towards the end of pregnancy.

Dr Berry Hart, in reply, said he had nothing further to say, and apparently had given the Society enough trouble; but the question was a very interesting one and must be treated in a scientific way.

With regard to Professor Robinson's investigation, he understood Dr Johnstone to say that if a number of ferrets were inseminated on the same day and killed soon afterwards, the embryos would be found in different stages of development; but if allowed to go on, the births would happen on the same day.

Dr Johnstone said practically on the same day.

Dr Berry Hart said that was nothing; the insemination periods in ferrets would vary, just as in cattle and sheep.

Meeting IV.—February 11, 1914.

Sir Halliday Croom, *President, in the Chair.*

I. The following gentlemen were elected Ordinary Fellows of the Society:—Patrick A. Bennet Clark, M.B., Ch.B., 15 Douglas Crescent, Edinburgh; J. H. Graham Hunter, M.B., Ch.B., Royal Maternity Hospital, Edinburgh; John L. Annan, M.B., Ch.B., 27 Braid Road, Edinburgh; T. Douglas Inch, M.B., Ch.B., Stobsmills House, Gorebridge; J. W. Pell, L.R.C.S. & P. Edin., 45 Merchiston Crescent, Edinburgh; Ernest Muir, M.D. Edin., 17 Craiglea Drive, Edinburgh

II. *The President* showed — TUBAL GESTATION AT THE EIGHTH MONTH. The specimen was interesting because of the fact that the patient had no symptoms whatever.

III. *Dr Haultain* showed—(*a*) ENDOTHELIOMA OF THE CERVIX, removed from a young woman, æt. 32. The only symptoms complained of were a great deal of pain in the back, with slight increase of menstruation—slight menorrhagia. On examination, a rugged, irregular condition of the cervix was found, simulating carcinoma, but on further examination it proved to be endothelioma of the cervix. The organ was removed by a modified vaginal operation in the usual way. So far there had been no recurrence. In a previous case in which an endothelioma of the body of the uterus was removed, the condition recurred within three or four months. In the present case he thought one might expect that there would not be a recurrence. Endothelioma was not so malignant as carcinoma, and certainly not so in a young woman of thirty-two. (A slide illustrative of the microscopical appearances was shown.) Endothelioma was extremely rare. Dr Donald of Manchester had had a case.

(*b*) RARE FORM OF CERVICAL FIBROID, interesting because of its rarity. The peculiarity was that the attenuation of the cervix was lateral. In his experience of at least forty other cases either the anterior or posterior lip had been the seat of the attenuation, whereas in the present case it was entirely confined to the lateral side of the cervix.

(*c*) STRANGULATED FIBROMYOMA FROM UTERINE TORSION (four times) The specimen was removed from a patient æt. 62. Nine or ten years after menstruation had ceased the patient began to suffer from a considerable amount of pain in the abdomen. The pain continued, not very severe but sufficiently so to require her to go to bed occasionally, for two and a half years; it then became worse, and signs of peritonitis appeared. Dr Haultain attempted to remove the tumour, and, in doing so, found it was subjected to considerable strangulation; it was engorged with blood below; the uterus also was in a similar state of congestion. The cervix was attenuated to the size of a lead-pencil, and measured about 4½ ins. in length. The uterus was twisted on itself four, possibly five, times. The patient had evidently for two and a half years before been suffering from a slow torsion of her uterus, until it became so great that it caused complete congestion and blocking of the veins, the pedicle being formed entirely of the cervix uteri. Such a condition of torsion was rare, and seemed to be worthy of bringing before the Society.

IV. *Dr Lamond Lackie* showed—RUPTURED UTERUS REMOVED from a patient in the Maternity Hospital about a week previously. The patient was a iii-para, this being her fourth pregnancy which had advanced to full time. The midwife had been called at 11 o'clock in the morning, and he believed she admitted having attempted to bring away the child; she failed, and sent for the doctor at 3 o'clock in the afternoon. Dr Lackie saw her at 5 o'clock; she was sent to the Maternity Hospital at

8 o'clock. She was not very collapsed, the pulse was fair, the temperature normal, and she was not extremely ill. On examination of the abdomen, the head was seen lying on the left side. There was no dulness in the flanks to suggest any bleeding into the cavity. The obvious thing was to bring away the child, and it was consequently decapitated. The question of rupture had then to be considered, and on examination a tremendous tear was found extending from one side right across to the other. The uterus seemed practically separated from the vaginal wall. At one point it was very difficult to say whether the tear was complete or incomplete. The difficult point was to decide whether one was in the abdominal cavity or not; unless the intestine was felt, it must be extremely difficult to say whether one was into the cavity or not. It was decided to remove the uterus, and the abdomen was opened. Very little blood was found in the abdominal cavity, and what there was was coming from the broad ligament. A supravaginal hysterectomy was performed. The patient was acutely septic at the time of the operation; the temperature rose to 102, and twelve hours after she died. He did not know whether the result would have been better from packing. He would like to hear the views of the Fellows as to knowing when the hand was introduced into the abdominal cavity or not.

Dr Jardine had never had to deal with a case where the hand went right into the abdominal cavity.

The President thought it was impossible to be quite sure.

Dr Lackie agreed with the President.

V. *Mr E. Scott Carmichael* showed — UMBILICAL CYST ADHERENT TO THE UTERUS. The patient was sent from the country to the Women's Hospital with a gastric ventral hernia. On making the usual section to expose the sac, and on opening the sac, he was puzzled to find he came upon a second sac. Further examination showed that he had opened into a loose

bag of fluid which was found to be adhering to the cæcum and the ascending colon, from which it had passed directly down to the pelvis, and was adhering to the tip of the uterus. After separation from the sac, the uterus and tubes were perfectly normal. The specimen was an unusual and interesting one.

VI. NOTE ON A CASE INVOLVING SOME ETHICAL QUESTIONS, EUGENIC AND OBSTETRIC.

By Dr J. Haig Ferguson.

X. Y. is an unmarried girl of 18, living with her parents, weak and facile in character, and about seven and a half months pregnant. She is fairly well developed physically, her appearance is by no means unattractive, but there is no doubt that she comes under the class now recognised as "degenerates."

She has a small pelvis: the diagonal conjugate measures not more than $3\frac{1}{4}$ ins., possibly only 3, which makes the true conjugate about $2\frac{1}{2}$ ins. or thereby. The pelvis seems uniformly contracted.

She is past the time for any thought of induction, with a pelvis such as hers, and the only possible treatment is of course Cæsarean section at or near full time.

It is in this connection that the case opens up some very interesting legal and eugenic points.

From a eugenic point of view it is probably advisable to sterilise this girl at the time of the Cæsarean section, but of course this cannot be done without consent being obtained. If the girl herself is asked and consents (*her* consent would not of course be given for eugenic or social reasons), then the knowledge that there is no further risk of pregnancy in a girl of her type makes it probable that she will become one of the greatest social evils in the community where she resides, where, the

doctor tells me, gonorrhœa and syphilis are practically unknown, and the fear of conception is the only factor which controls promiscuous intercourse among the young people of the district.

From a medical point of view it is not usual to sterilise a woman nowadays in her first Cæsarean section (in the absence of other disease), however small her pelvis may be.

The first child may not survive, or may be weak or deformed, and it is right, now that Cæsarean sections are so comparatively safe, to give the woman at least one more chance should she marry.

Apart from the social difficulty in a case like this, there comes another question. Supposing one were to sterilise this patient without getting her consent, and supposing she were to marry later, and by some means it were discovered that this had been done, no matter how good the reason may have seemed to be for it, there might be good claim for legal damages against the operator.

The parents of this girl have been consulted on the question, and the whole matter has been explained to them. I understand that they distinctly wish their daughter to be sterilised. They propose to sign an authorisation to the operator granting their full consent to sterilisation, though I am by no means quite sure that this would be an absolute legal safeguard in dealing with a girl of her age.

The parents do not wish the girl to know that she will be unable to conceive again, and still less do they wish this to be a matter of general knowledge in the district.

It may be said that we as medical men have nothing to do with the social or moral aspects which bear upon a case of this kind, and in a sense this is quite true. But, on the other hand, we should endeavour to do nothing, however justifiable it may be in itself medically, which is likely to bring difficulties into any community.

The knowledge by an unmarried woman that she cannot

conceive may lead to disastrous results in many ways. I have known one instance where (after the removal of two ovarian dermoids) it completely demoralised a girl, at one time of the highest character, who ultimately became syphilitic.

Another case is recorded where a girl who had become syphilised, vowed vengeance on the male sex generally, by doing her best to infect as many men as possible (500 being her ambition)—truly a dangerous woman in any community!

I shall be glad to hear the views of the Fellows of the Society on this case which I have shortly summarised. It is undoubtedly an unusual one, and as it has very practical bearings, I hope it may lead to some discussion.

Dr Berry Hart said Dr Haig Ferguson had raised a great many interesting points. Firstly, what should be done in this particular case? The right thing was to do a Cæsarean section and leave the girl as she was. The legal difficulties had also been pointed out. But this case was merely nibbling at the whole question. The care of the illegitimate child was a thing that was neglected tremendously in this country, and the fate of such a child was often a very hard one. In this country nothing was done; abroad there were honorary guardians appointed, and the father had to pay a certain amount. Dr Berry Hart had known some very dreadful cases, where even the illegitimate children of people in good society were allowed, after the age of 14, to drift into the very lowest class of society. The question of deformities of the pelvis was also raised; these were due, to a great extent, to improper food, another thing which in this country was not looked after. The whole question, however, was so extensive one did not care to approach it on the basis of one case; statistics were required.

Dr Haultain was interested in the case because of its having come under his care at the Maternity Hospital about

a week ago. The patient on questioning seemed to him quite an intelligent girl, so far as her family connections were concerned. The parents had written him saying he could do anything he liked with her, sterilise her or anything. On admission the patient was asked what her desire would be, but she absolutely refused to give any decision in the matter whatever. Well, naturally, being somewhat canny, and keeping the prospect of severe damages in the future in view, he decided to perform Cæsarean section and leave the condition otherwise *in statu quo*. This was done four or five days ago. In coming to this decision he considered very thoroughly first of all the question of what the result of removal of the ovaries might be. If she lived, it was quite conceivable that she might become worse in her habits and give rise to very considerable disease in the community. On the other hand, he thought, it was quite possible that if she had a child she was not so degenerate in her mind but that it might have a salutary effect upon her. It was quite possible that the time would come when the girl might improve in her nature, might marry and become quite a respectable citizen. From that point of view it seemed to him the right thing to do was to leave her as she was. The other point of view was by leaving her as she was, would she go on continuing to beget illegitimate children? From the point of view of the children it seemed rather hard. But still, taking everything into consideration, he felt that there was no reason for him going further than he had done.

Dr Lamond Lackie said the case was very interesting because such problems might occur to all men engaged in hospital practice. He related an experience which he had had two years previously in the Maternity Hospital. A patient was admitted in the second stage of labour who had had a previous craniotomy performed. The first child was illegitimate, as was the second. The c. v. was $3\frac{3}{4}$ ins.; forceps failed, and

pubiotomy suggested itself. Accordingly Dr Lackie suggested to the patient that he should perform a simple operation which would enable her to have a living child. She, however, wished the child destroyed, and the same operation done as on the previous occasion. Craniotomy was therefore performed, but he wondered if one was justified in doing craniotomy where the risk was just as great as in pubiotomy, and whether one was justified in destroying what might become a useful citizen. At all events he did destroy the child, and he would be interested to hear what the members thought it was right to do under these circumstances. Personally he thought one had no right to do craniotomy if pubiotomy had no greater mortality than craniotomy. But the patient would not have it; she wished the child destroyed; it suited her best.

Professor Jardine (Glasgow) thought that in the present case the proper thing had been done. So far as the illegitimate cases he had to deal with were concerned, he never sterilised unless there was some other good reason for doing so. If the woman marries afterwards, she ought to have a chance of having children. As regards repeated Cæsarean section, if a woman came to him for Cæsarean section for the second time, he sterilised her. It might be said, why not let her go on? Recently he had a case in which the operation was performed for the third time; he himself had done it first, then another man had done it and had refused to sterilise. When the patient was admitted, the child was felt so distinctly through the thinned uterine wall that it was evident that if the woman was left for any length of time, the uterus would rupture. They had had cases in the Maternity Hospital where the wall had actually given way, so that the uterus was not in a very safe condition after the operation had been done. He thought the condition might be left till the second pregnancy, but after that the risks were great. If the patient or her husband would

not have it done even then, it was not of course done; but usually if they are given the option, they jumped at it after the first Cæsarean section. In this case the girl was better left as she was, and, as Dr Haultain said, the presence of the child might help to bring her to her senses and make a better woman of her.

Such a case as Dr Lackie's he had not been called on to deal with, but in dealing with these patients he told them he was going to do an operation, and did it. He had done craniotomy where it was the best thing for the mother; but if one were brought face to face with a case where one had to act under the circumstances in Dr Lackie's case, one would be very much inclined not to do it.

Dr W. Fordyce mentioned a case with which Dr Barbour and he had had to deal in the Maternity Hospital. The patient was a very degenerate woman, the most degenerate he had ever seen; at that time she was not married, but was three and a half months pregnant, with a narrow pelvis. She was sent to the Maternity Hospital to have abortion induced by her own wish, her mother's wish, and the doctor's command. This was done, although personally he thought it should not have been done, as probably the patient would become pregnant again. She did, and came back eight and a half months or nine months pregnant, but a respectable married woman. Then the question arose, was the woman to be sterilised at the time of the Cæsarean section? The patient and her mother refused permission. Unless one got the consent of the patient, he could not imagine any conditions which justified one in sterilising a patient; at anyrate, one rendered oneself liable to an action for damages. If there was any other disease, of course, that was quite different.

As regards Dr Lackie's case, which was a much more difficult one, the mistake was, of course, in asking the patient

at all; Dr Lackie should have said he was going to do an operation which was safer than the one which had been done before.

Dr Barbour said he recollected the case referred to by Dr Fordyce, and the discussion about it. He (Dr Barbour) was not sure but that the after-results had justified the course adopted. In the first place, had the child gone on to full time and been delivered by Cæsarean section, the patient would have been handicapped by having an illegitimate child, which would probably have prevented her subsequent marriage.

Dr Jardine raised an extremely interesting point when he said that Dr Haultain had done the right thing in Dr Haig Ferguson's case. Dr Barbour supposed that was because the patient had not a contracted pelvis. On the other hand, when dealing with a married woman with a contracted pelvis he would sterilise her. Personally he did not think one should do on a married woman what one would not do on an unmarried one. He took it that if the patient quoted by Dr Haig Ferguson, in addition to the degeneracy had had a contracted pelvis, Dr Barbour would have said that the right thing had not been done and that she should have been sterilised. (It was here pointed out to Dr Barbour that the patient had a contracted pelvis.) Well, why should one do one thing in the case of a married woman and another in the case of an unmarried woman? By so doing one was giving the married woman fewer rights than the unmarried.

Dr Haig Ferguson had not made out a very strong point for sterilising the patient from the point of view of her moral degeneracy. One must be very careful about imposing laws and conditions on a patient. He agreed with Dr Haultain's view, and hoped that having a child would lead her to be a better woman.

Dr James Ritchie thought the case one of very great diffi-

culty, and that difficulty lay not in the fact of the patient being an unmarried woman, but a degenerate. In this country there was a tremendous increase of degenerates. In the upper classes the children used to number six, now it was two; whereas in the lower classes of the population the number was six. Dr Haultain had not found her to be so degenerate as he (Dr Ritchie) had gathered from Dr Haig Ferguson she was; she seemed to be a somewhat intelligent girl; and, as all knew, often a woman without any great amount of intelligence, given a healthy husband, brought forth a child who ultimately took a very good place in society. In this particular case he agreed that Dr Haultain did the right thing in not sterilising the patient.

In Dr Lackie's case the patient should not have been consulted at all, and he should have done a pubiotomy; he did not think Dr Lackie was justified in destroying a child simply because the mother wished it done.

Dr Lackie said he would have been assaulting her if he had not consulted her.

Dr Ritchie thought the obstetrician had to do what was best.

Dr Lackie remarked that one could not operate without consent.

Dr Berry Hart asked if Dr Lackie thought she would have brought an action against him for presenting her with a living child.

Dr Lackie replied that he thought so.

Dr Young thought the attitude Dr Lackie took up, that, if the risk to the mother was about equal in craniotomy and pubiotomy, he was justified in doing the former, was the right one. As a matter of fact, however, statistics showed that in the case of an aseptic woman, who had been fairly well looked

after in the early stages, the risk from craniotomy was practically nil; whereas the risk from pubiotomy, in which the conditions were similar, was at least 2 or 3 per cent. The risks in association with the impairment of the locomotor powers of the patient, which were not infrequently met with, had also to be remembered. Altogether, from the point of view of the mother, who after all must decide the question, he thought Dr Lackie did the very best thing. In a Court of Law, the mother's interests being paramount and receiving much more consideration than those of the child, pubiotomy would have raised a very serious question with regard to Dr Lackie's case.

Dr Davidson said, with regard to Dr Haig Ferguson's case, the whole question depended on whether the girl was mentally deficient or not; if she was, then the parents' consent would be sufficient. Dr Lackie in dealing with his case could do nothing else. It was the mother's own child, and she had the right to say whether she would have an operation to save the child or the one safer for herself.

The President agreed that the fate of the illegitimate child was a very bad one, and the less they had of them the better, when they could get any legitimate reason for preventing the birth of illegitimate children. He had performed Cæsarean section as often as three times. He thought Dr Haig Ferguson's suggestion of sterilising was a very appropriate one indeed. It was a question of whether the woman was really degenerate, as Dr Davidson had pointed out; if she was degenerate, whether one had permission or not, one ought to sterilise. He quite agreed with Dr Young's remarks regarding Dr Lackie's case.

Dr Haig Ferguson, in reply, thanked the Society for their kindly reception of the paper, and for the remarks made. He

quite agreed that Dr Haultain had done the best, assuming that the woman was not degenerate. He (Dr Haig Ferguson) had not full information on that point, because what he got was second-hand. The question of sterilisation was not raised on account of the smallness of the pelvis. With regard to the question of degeneracy, he had consulted Sir Thomas Clouston, who had written him to the following effect:—"From my point of view the first thing that occurs to one is, Does X. Y. come under the new 'Mental Deficiency and Lunacy (Scotland) Act'? It is difficult without a personal examination to give a reliable opinion on the point, but the presumption is that she does. In that Act 'Feeble-minded persons' and 'Moral imbeciles' are thus defined—Feeble-minded persons, that is to say, persons in whose case there exists from birth or from an early age mental defectiveness not amounting to imbecility yet so pronounced that they require care, supervision, and control for their own protection or for the protection of others.

"Moral imbeciles, that is to say, persons who from an early age display some permanent mental defect, coupled with strong vicious or criminal propensities, on which punishment has had little or no deterrent effect. Probably X. Y. has undergone 'punishment' from her parents. If she comes under either of these two clauses, her parents' consent to sterilisation would be valid against a legal action. Even if it is doubtful whether she comes under that Act, it seems to me—but my opinion on this point is not worth more than anyone else's—that a surgeon is abundantly justified in sterilising her as a part of the Cæsarean section, on the ground of prevention of any risk of another pregnancy and the impossibility of natural delivery. The risks of what would happen to a sterilised girl going about at large are so purely ethical and social that a surgeon has practically nothing to do with them."

VII. ADVANCED EXTRA-UTERINE PREGNANCY WITH TUBERCULOUS PERITONITIS.

By Professor Sir Halliday Croom.

It seems an anachronism to report a case of advanced extra-uterine pregnancy, undiagnosed and unoperated upon. Such was the common history of extra-uterine pregnancies some decades ago, when they were more or less pathological curiosities. But nowadays one has to apologise for reading a report of such a case, because at the present time extra-uterine pregnancy is a distinct and complete gynæcological entity.

It is a matter of fact that many early extra-uterine pregnancies are overlooked, and in many the symptoms are so slight that they attract no attention, and may end either as a mole, or an abortion. But I have not, in my experience, met with a case of advanced extra-uterine pregnancy which has escaped notice altogether, which has given rise to no symptoms of any kind, and which, as the records of this case show, must have been a tubal pregnancy which had ruptured and which had become re-encysted.

At the present time the accidental discovery in the post-mortem room of an extra-uterine pregnancy, which has not been previously diagnosed, is, and must be, a comparatively rare occurrence.

The patient was admitted to my wards in the hospital six months pregnant, with the complication of tuberculous peritonitis. Owing to the distended condition of her abdomen, careful examination either abdominally or *per vaginam* was very difficult.

She never had, at any period of her pregnancy, any sign or symptom of an ectopic gestation.

She was admitted of course, because she was pregnant, and except that her uterus appeared to be larger than corresponded

with the menstrual period, there was no other apparent abnormality in the pregnancy.

The case is as follows:—

Mrs W., æt. 32, primigravida. Sent into the Maternity Hospital on 30th October 1913, by Dr Scott, Broxburn.

The patient had been married for eight years, but had never been pregnant before. She stated that her last period occurred some time in July 1913.

History of present condition.—For the two months preceding admission the patient had not been feeling well, and had required to take a great deal of rest. This she put down to her being pregnant. Five weeks before admission she noticed that her abdomen was increasing in size, and she thought that the rate of enlargement was too rapid to be accounted for by pregnancy. She therefore consulted Dr Scott, who discovered ascites and treated her for that. As no improvement resulted and the patient was growing daily weaker, he sent her to hospital, with the alternative diagnoses of hydramnios or concealed accidental hæmorrhage.

During the whole of her illness the patient had never experienced any pain. During the last three days before admission she had occasional attacks of vomiting, and the bowels had not moved.

State on admission.—The patient was very weak and emaciated. She was suffering no pain at all, but appeared rather collapsed. The temperature was 102° and the pulse rate 132.

Examination of the Abdomen.—On inspection the abdomen was enormously distended, the swelling reaching up to the ensiform cartilage, and causing some bulging in the flanks. Except for a few poorly marked striæ, the abdomen did not in appearance resemble that of a pregnant woman. On palpation there was no pain or tenderness anywhere. The abdominal swelling was neither hard nor tense, but soft and fluctuating. A tumour could be felt rising out of the pelvis and extending to the umbilicus. It was irregular in shape and occupied most of the transverse breadth of the abdomen. Even without an anæsthetic it could be moved about freely.

On percussion there was marked dulness in the flanks, which altered according to the position of the patient. In front the percussion note was tympanitic above the umbilicus and dull below it.

On auscultation no fœtal heart nor uterine souffle could be detected.

Vaginal examination.—The cervix was soft, but not so soft as usual at the supposed stage of pregnancy. It was slightly open, but not canalised, and what was taken to be a presenting part could be felt vaguely and indeterminately.

Bimanually, the tumour could be palpated, but not clearly, owing to the quantity of fluid in the abdomen. It felt like a six months' uterine pregnancy.

After examining the patient, I excluded both hydramnios and concealed accidental hæmorrhage, and came to the conclusion that the case was one of pregnancy with tuberculous peritonitis. Owing to the feeble condition of the patient, I did not think operation advisable. Dr Haig Ferguson, who also saw her, agreed with me in the diagnosis and as to the advisability of interference.

After the bowels had been emptied, the vomiting ceased and the temperature dropped to 100°, the pulse remaining at 130. She was placed upon strychnine hypodermically. Next day the temperature rose to 102 and remained about that level for five days, during which time the pulse dropped from 130 and 140 to about 110.

I asked my colleague, Professor Caird, to see the case, and he came to the conclusion that the condition was either tuberculous peritonitis or some obstruction to the portal circulation. He advised that nothing be done, but if the fluid increased markedly some of it should be withdrawn.

In the following days her temperature remained very irregular, and the pulse became increasingly weaker. Death ensued on 22nd November, twenty-three days after admission.

The following is the pathologist's account of the autopsy:—

The body is much emaciated, contrasting with the enormously distended abdomen. On opening the abdominal cavity,

a very large quantity of turbid brown fluid escaped, which had no smell. Adhesions were present between the liver and spleen and the parietes, and between the coils of intestine. The sigmoid colon and omentum were adherent to the uterus. The peritoneum was generally covered with minute yellow projections. There was some enlargement of the mesenteric and retro-peritoneal glands. The liver was normal in size, but showed chronic perihepatitis with adhesions to the diaphragm. On section it showed well-marked fatty change, particularly at the periphery of the lobules, the central zones being congested. The spleen was greatly enlarged and adherent to the diaphragm. On section it was dark red in colour and firm in consistence. Numerous minute white areas were scattered throughout it.

Both kidneys were enlarged and pale, showing on section a swelling of the cortex. In the right kidney there were numerous scattered pale areas, probably tuberculous. The suprarenals were normal.

The uterus was enormously enlarged, and presented a ragged opening on the left side posteriorly, through which the foot of a fœtus projected. Both lungs showed numerous adhesions to the parietal pleura and numerous scattered grey and yellow areas.

The diagnosis therefore was tuberculous peritonitis with rupture of the uterus, tuberculosis of the kidney and of the lungs.

The pelvic organs were removed *en masse* and further examined by Dr R. W. Johnstone, when it was discovered that the pregnancy was not intra- but extra-uterine. The following is Dr Johnstone's report on the specimen :—

"The specimen consists of the pelvic organs with the pelvic colon and some adherent omentum. The whole mass is about the size of an eight or nine months' pregnancy. On dissecting away some of the adhesions, the uterus is found lying below and to the right of the gestation sac. It is enlarged, the cavity measuring $4\frac{1}{2}$ ins. by the sound. The endometrium is

distinctly thickened. The right tube and ovary can be distinguished, although both are surrounded by adhesions. The right tube is very slightly distended, with what on section looks like mucopurulent material. The gestation sac is distinct from the uterus and occupies the position of the left tube. The left ovary can be seen matted down to the surface of the tumour. The rectum, the pelvic colon and its mesentery, with the left broad ligament, are spread over the posterior and upper aspects of the tumour, to which they are densely adherent. Posteriorly and on the left side of the tumour there is a ragged opening through which the foot of a fœtus is projecting. Except where other organs are adherent to it, the sac wall is for the most part no thicker than blotting-paper. (Fig. 1).

"On opening it the sac was found to contain a mature fœtus, lying more or less transversely with the cord twisted round its body. The placenta was attached inferiorly and posteriorly, and the rectum is adherent on the outside over a great part of the placental site. Several large vessels are present in the wall in the neighbourhood of the placenta. The placenta itself is somewhat kidney-shaped, and measures 20 cm. (8 ins.) long by 12 cm. ($4\frac{1}{2}$ ins.) broad. At the middle it is actually 8 cm. ($3\frac{1}{4}$ ins.) thick. The cord arises near the centre and runs over the surface, bound down to it by amnion, to the convex edge. Here it forms a small projecting loop which is also bound down by amnion, before it passes off to the fœtus.

"The sac wall appears to consist of a thin layer of rather structureless tissue, which, after hardening, is extremely brittle. There is apparently no peritoneum over it except where the bowel or omentum is adherent.

"Microscopic sections were made of (*a*) the uterine wall, (*b*) the placenta, (*c*) the sac wall, and (*d*) the right tube.

"The uterine wall showed that the endometrium had become converted into a decidua, which showed no abnormal characters. The sections of the placenta also showed little abnormality, except the absence of the decidua. At the maternal surface the villi pass through a thick layer of fibrinoid material, and are attached to it and to the underlying connective tissue.

[*Face page* 150.

FIG. 1. Posterior view of uterus and gestation sac. A sound has been passed into the uterus, which has also been opened above.

Nothing in the shape of a decidua was seen in the sections examined.

"The sac wall was found to consist mainly of several layers of apparently organised fibrin on the outer side, while inside there were areas of connective tissue with blood-vessels. There was in parts a very considerable infiltration of leucocytes. At the part examined peritoneum could not be recognised as part of the sac wall. The most interesting point is, however, that at certain points large cells like decidual cells may be seen—oval or rounded in shape, with large pale nuclei. Apparently this is an attempt at a decidual reaction on the part of the connective tissue cells. The cells are certainly not like trophodermic cells, and the section was made from a part of the sac far removed from the original site of the tube.

"The sections of the right tube showed that the lumen was distended and full of a mass of leucocytes and fibrin. The folds of the mucosa were almost entirely obliterated, and there was in parts a very considerable increase in the subepithelial stroma. The epithelial cells are greatly swollen, and in some parts appear to have degenerated into a true syncytium or symplasma The underlying stroma cells are also rather enlarged, but nowhere was there any appearance of a real decidual reaction. Nowhere was there any sign of tuberculosis.

"Apparently the original implantation of the ovum had been on the inferior and posterior aspect of the tube. From the size of the fœtus this must have occurred at least four months before the cessation of menstruation in July. Furthermore, rupture of the tube must have occurred quietly and practically without symptoms.

"It seems possible that the dislocation of parts that must have occurred when rupture took place, may have been the cause of the strange loop of umbilical cord at its attachment to the edge of the placenta."

Dr W. Fordyce had seen a similar case in Manchester in a patient who had menstruated eleven months previously; she had an enormous abdomen; Dr Fothergill operated on her. A

number of physicians saw her and scouted the idea of pregnancy; it was thought to be an extra-peritoneal ovarian cyst; but on opening the abdomen, it was found to be an ectopic gestation. The head of the child came out of the opening; the fœtus was a very large one and had been dead for some time; it had gone to full time and died, but there were no symptoms.

Dr Barbour thought the paper extremely interesting. Extra-uterine gestation in a detached form might, of course, be absolutely symptomless. In a paper read some years ago Dr Angus Macdonald (senior) recorded a case in which he operated in the belief that there was a fibroid, but on cutting through the neck of the fibroid he came upon a child. That pregnancy had gone to full time, and the patient had had no symptoms.

Dr Haig Ferguson said the interesting point was the fact of the patient being tuberculous. Personally he did not think there was any pregnancy at all, but that it was only an ordinary peritonitis. The patient had amenorrhœa, which one expected in tuberculous disease.

Dr Haultain said that a few years ago he had shown a fœtus to the Society from a patient who many years before had thought that she had a pregnancy. It passed off, and everybody laughed at her. The tumour remained but got smaller, and she had it for forty-two years. When she died at the age of 67 the fœtus was found.

Meeting V.—March 11, 1914.

Sir Halliday Croom, *President, in the Chair.*

I. THE ÆTIOLOGY OF ECLAMPSIA AND ALBUMINURIA AND THEIR RELATION TO ACCIDENTAL HÆMORRHAGE (An Anatomical and Experimental Investigation).

By James Young, M.D., F.R.C.S.

The study of eclampsia (and the albuminuria of pregnancy) can be carried out from two points of view, the *clinical* and *anatomical*, which concerns itself with a description and interpretation of the symptoms and the morbid changes present, and the *experimental*, in which an attempt is made to reproduce these symptoms and morbid changes in the lower animals. The investigations here recorded were, to begin with, carried out along clinico-anatomical lines. From these preliminary studies there soon emerged an experimental line of inquiry. As a matter of fact, this was entered upon as a necessary test of the validity of the conclusions to which the preliminary investigations had led.

Within recent years most of the investigations into the cause of eclampsia have issued from the belief that the placenta is the most likely source of a poison, which all are convinced is the cause of the disease. There can be no doubt that pregnancy is the cause of eclampsia and that the poison is, at anyrate in its ultimate origin, to be traced to the child or to the placenta. That it is not the child is proved by its occurring in cases of hydatid mole, where there is no fœtus. This leads inevitably to the conclusion that the direct or indirect source of the poison resides in the chorionic elements. That the relationship between the uterine contents and the

toxæmic states is a very direct one is shown by their frequent immediate cessation after the emptying of the uterus, or after the intra-uterine death of the fœtus. On the other hand, even when the child dies *in utero*, the toxæmia may persist until the child and placenta are delivered. It would in such cases seem clear that the death of the child (the macerated state of which at delivery testifies to the time when this death occurred) must, by the consequent thrombosis of the vessels, prevent any poison which might have been produced in it from being conveyed to the placenta. The placenta, therefore, in such a case, must be the source of the poison; and to my mind, the only interpretation legitimate under such circumstances is that, although the placenta usually becomes detached from the uterine wall when the child dies, it may in some cases remain fixed. When this is so, it will continue to pour into the blood-stream the toxic materials, and thus perpetuate the condition which killed the infant. We thus see that an analysis of apparently irregular and irreconcilable data leads to a simple explanation.

It is impossible to gainsay the fact that there is an intimate and perfectly direct association between the uterine contents and the toxæmia. One fact, however, apparently inconsistent with such a view, is *post-partum* eclampsia. In about 20 per cent. of cases eclampsia occurs after labour. The great majority occur on the first day, and probably within a few hours after labour. No matter how we would explain these cases, we must inevitably start by admitting that something of a chemical nature has been left behind after the birth of the child and the placenta. We thus see that any experimental investigation into the cause of eclampsia must concern itself primarily with the attempt to isolate from the placenta (not from the child) some chemical substance that can reproduce the changes in animals.

Relation between Albuminuria and Eclampsia and Placental Disease.

It has for long been recognised that there exists a definite association between the toxæmic states and placental disease. On the other hand, infarcts of the placenta are not infrequent in cases where there is no sign of toxæmia. They are more likely to be present in the last two months of pregnancy; and Eden has suggested, with great reason, that they are to be looked upon as signs of senility in a short-lived organ. Their relative frequency has been differently stated by different authors. Meyer found them in 2 per cent. of 1124 placentæ, Rossier in 17½ per cent. of 1174, whilst Whitridge Williams found white infarcts, measuring 1 cm. or more in diameter, in 63 per cent. of 500 placentæ. Such figures, though exhibiting a wide margin of difference, leave no doubt regarding the relatively high frequency of the condition. On the other hand, there can be equally little doubt that these changes are associated in some special manner with the toxæmias, albuminuria and eclampsia. In Rossier's figures given above, 54 of the women suffered from albuminuria, and, in these, infarcts were three times more common than where the urine was healthy. In Meyer's patients, where albumin and casts were present, infarcts were four times more frequent than where there was a normal urine. Fehling found them in 50 out of 91 albuminuric patients. "It is accordingly apparent," says Whitridge Williams, after stating these figures, "that the majority of investigators, who have busied themselves with the subject believe that a marked relation exists between albuminuria on the part of the mother and infarct formation in the placenta." The infarcts which are of greatest importance in this respect are those of the recent or red variety. It is with them that albuminuria is especially associated. White infarcts, as such, seem to possess little clinical

importance. Out of 7 cases of eclamptic placentæ examined by myself, there were massive red or purple infarcts in 5; whilst out of 6 cases of severe albuminuria, there were similar structures in 3. That is, in 13 toxæmic placentæ there was recent placental disease, evident to the naked eye, in 8—*i.e.*, roughly 60 per cent.

"What is the meaning of this obvious association between placental infarction and the toxæmias?" is a question that has often been asked. Most workers admit that it is impossible to explain this relationship satisfactorily, or they attribute it to the action of the general toxæmia, of which the albuminuria is an evidence. For example, Eardley Holland, whilst admitting that infarcts are more frequent in albuminuria and eclampsia, says that "they may be looked upon as the result of a chronic toxæmia; as to their connection with eclampsia, they are merely accompaniments, not consequences. The presence of these chronic degenerative changes in eclamptic placentæ has been investigated by Brindeau and Nattan-Larrier, who give them no special significance." Whitridge Williams, in a summary of the position, in so far as he could interpret it, said "marked infarct formation is not infrequently observed, and often results in the death or imperfect development of the fœtus. It is usually associated with albuminuria on the part of the mother, though at present we cannot account satisfactorily for the relation between them."

Placental disease of this kind has been usually looked upon merely as an accompaniment or as the result of the toxæmic state. So far as I know, no serious attempt has been made to regard the placental disease as the cause of the toxæmia, and this in spite of the fact that there is, in all the evidence which has accumulated through the years, not one single item which is logically inconsistent with such an interpretation.

It *is* true, then, that placental infarction may be present without any evident toxæmia. It is also true that there may

be a severe toxic condition, and no evident change in the placenta. It is the very readiness with which the truth of these statements can be proved that has, in my belief, obscured the real nature of the relationship. To complete the statement of this relationship, where there is massive recent infarction there is always a toxæmia.

The meaning of this apparent paradox will become apparent when I say that, after I had been working at the subject for some time, studying the placentæ in their clinical relationships, I discovered that, *if there is an acute toxæmia ending rapidly in labour, the placenta may present evidence of disease, or it may look perfectly normal to the naked eye. If, on the other hand, the acute attack passes off and labour only supervenes, say ten or fourteen days after, we find extensive recent necrosis. Plate I., Fig. 2, is a good example of such a case. This important finding at once suggested that it is the recent autolytic changes in the affected organ that generate the poison.* As I shall show, this conclusion from anatomical data is completely supported by the experimental results. It is this that explains why, in an albuminuria which becomes established gradually and persists for some time, one is more likely to find marked placental disease. *It is just the comparatively slow involvement of the placenta that allows of the continuance of the pregnancy and the evolution of the infarcted regions. Where there is a sudden and extensive involvement of the placenta, the toxæmia is so fulminant that the pregnancy ends before any naked-eye changes in the placenta are produced.* The import of these statements will emerge in a clearer light later when the experimental work is entered upon.

It should be remembered that the placental organ is unique in the sense that, if a part of its substance undergoes necrotic changes, the degenerating patch is a massive poison focus bathed all round by circulating maternal blood. A ready access into the systemic circulation of any toxic material is thus

allowed. There is no other region in the body where such a condition of affairs could obtain.

It is easy to understand that a comparatively small placental disease may occur without any untoward effect. Under these circumstances the absorption of toxic products from the dying patches is so gradual, and at any one time so small in amount, that it is tolerated. Where, however, this normal and limited change becomes excessive there is a pouring into the maternal circulation of more poison than the system can safely deal with. Such an explanation accounts at once for the presence of the toxæmia, where the placental disease is marked. I contend that it is a much more logical one than to suppose that the toxæmia develops independently and acts upon the placenta by hurrying up and exaggerating a process that is so common in a smaller degree as to be almost normal. The one view is an explanation of a relationship, the other is meaningless, except as the statement of an extraordinary coincidence.

Mild degrees of albuminuria are common in pregnancy, and may well be due to those cases of comparatively small placental involvement. An explanation, on the lines I have laid down, accounts likewise for the chief time incidence of the toxæmic states we are considering being in the last two months of pregnancy, just the time when the senile changes in the placenta are most likely to be in evidence. It accounts also for the very frequent cessation of the symptoms with the intra-uterine death of the child and the detachment of the placenta. This circumstance is due to the fact that the separation of the placenta withdraws the poison-laden foci from the maternal blood-stream, and the protective mechanism thus initiated is continued by the blocking of the open mouths of the maternal vessels by the thrombosis that quickly occurs.

The Cause of Placental Infarction.

I. *General Anatomical Considerations.*

The placenta is a temporary structure, and is so constructed that it can come away readily during labour. It is only so closely incorporated with the uterine wall that shearing on this wall is prevented. As a matter of fact, naked-eye examination shows that all that serves to bind placenta to uterus are a number of thin films of decidua. As I have shown in another place, there are, in the uterine mucosa, none, or practically none, of the supporting elements, such as muscle, elastic tissue, etc., which are found in other regions. Derived as it is from the mucosa, the same applies to the decidua. The vessels lose these supporting coats as soon as, or nearly as soon as, they enter the placenta. This fact must also be considered in relation to the loose connection which is necessarily present between placenta and uterine wall.

If we examine the placenta still attached to the uterine wall, it is found that the thin decidual films, which attach the placenta, bridge across large venous channels. In point of fact, the placenta seems to rest almost entirely on a greatly expanded blood lake, broken up into individual spaces by the bridging sheets. Even to the naked eye one can see these spaces opening on the one side through the placental surface into the intervillous space, and, on the other side, becoming continuous with the sinuses in the muscular wall of the uterus. On the placental side the blood spaces are lined by a thin layer of compact decidua. As we know, from microscopic examination, this thin layer is complete practically all over the placental surface. Where the vessels perforate the placenta it is absent, and, in addition, here and there, comparatively large tracts on the surface may be seen where the villi dip straight into the blood spaces. It is through such openings that Veit believes

there may be a wholesale "deportation" of villi into the maternal blood-stream. The separation of the placenta is rendered easier by an interesting change which occurs in the decidua. This consists of an œdematous loosening of the tissues, and is often seen in separated placentæ just under the compact layer (Fig. 1), as also in the decidual plugs that extend from this layer into the substance of the placenta.

This arrangement between placenta and uterus is ideal from the point of view of ease of separation, but it obviously also means a ready tendency to vascular disturbance. There is a very loose organic binding, and to all intents and purposes the placenta merely rests against the uterine wall. When the child is expelled the retracting uterus grasps and expels the placenta, the soft decidual fixing strands offering practically no obstacle. During pregnancy there is nothing to disturb the placental fixation. It is known that the uterus contracts intermittently, but this can do nothing but empty the placenta of its blood. The liquor amnii will disseminate the tension equally over the uterine and placental surfaces, and it is likely that the constantly recurring intermittent uterine contraction, by expressing the placenta at definite intervals, leads to a heart-like, pumping action. The placental blood space is, in a sense, a greatly expanded maternal vessel. When a decidual vessel pours its blood into this a great diminution in velocity and force must take place, just as occurs when a river pours its waters into a lake. This circulating speed will become less and less as the small stream issuing from the vessel passes further and further into the placental blood lake and becomes wider and wider. The flowing force, therefore, in this space, derived from the *vis a tergo* of the general circulation, must be extremely low, and for an efficient passage of the maternal blood throughout and away from the placenta other factors must come into play. The recurring systole and diastole of the uterine muscle must, by exercising respectively a compression

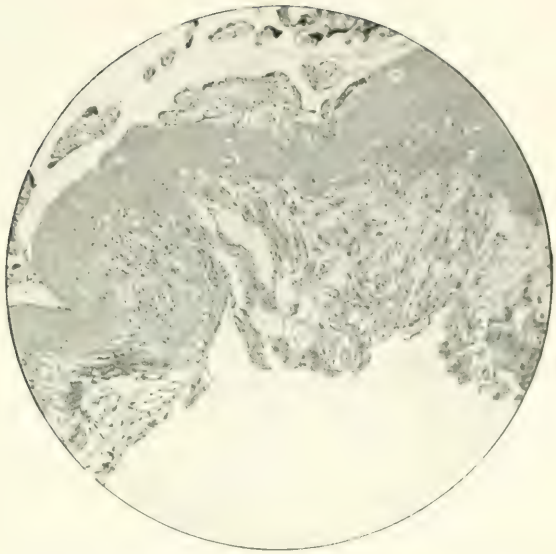


FIG. 1.—Decidual surface of placenta. Villi are seen above, then the thin compact decidua, and under this the loose decidua at the line of cleavage.

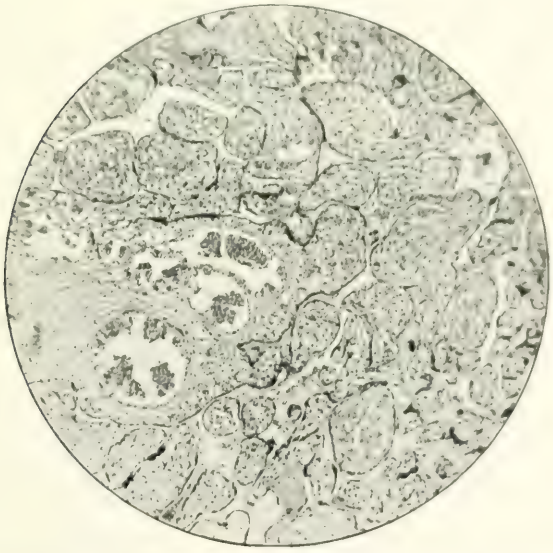


FIG. 2.—Recent (red) infarct. Villi packed together. Note the engorgement of the vessels. Case of accidental Hæmorrhage without Albuminuria.

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and a suction action, play an important part. We can imaginatively recognise the necessary existence of other factors. Thus, the villi are greedily absorbing fluid from the circumambient blood, and this must entail a continual mass movement of the blood in the space. Then, the taking up of oxygen and the other ingredients in solution in the blood, and the giving out of waste products, must mean a constant chemical alteration in the blood with constant diffusion currents backwards and forwards of the respective chemical ingredients in an effort to readjust the equilibrium.

II. *Changes Present in Localised Placental Death.*

Every person who has studied even a small number of placentæ is familiar with the so-called white and red infarcts. These consist of cubical, wedge-shaped, or irregular, sometimes sprawling, areas of yellowish or reddish tissue, which have lost the normal spongy appearance. They are firm in consistence. In the great majority of cases they lie with their base against the decidual surface. Sometimes an infarct may, at first sight, seem to lack this relation to the decidua, and to be embedded in the centre of the placenta or even to lie up against the chorion. In the majority of cases, however, on tracing the sections of the infarct, it is found to strike the decidua at a broad base. The oblique position of the infarcted region is what led to the erroneous first impression. In my experience, this decidual relation is so constant that it clearly must be considered as of great value and importance in any attempt to throw light on the causation of the condition. Many infarcts of the red or purple variety are difficult or impossible to recognise in the fresh placenta. When the placenta is put into fixing solution the blood quickly oozes from it, because in the healthy intervillous spaces it does not clot. (This it is which explains how rarely we find the villi embedded in blood in microscopic sec-

tions of placenta.) With the escape of blood the cut surface of the placenta, where it is healthy, becomes much paler. The diseased areas remain dark, and are thus thrown into relief against the paler background.

One fact which has emerged clearly from my investigations is that the various appearances included under the term "infarct" are different stages in one and the same process, and not, as is often stated in the text-books, independent pathological states.

(1) In the earliest stage recognisable to the naked eye the patch is deep red, purple, or even black. *It may not be visible till thrown into relief against the surrounding paler placenta, which has lain for some days in the fixing solution, into which the unclotted blood in the intervillous spaces has oozed.* This stage is seen in Plate I., Figs. 1 and 2, and Plate II., Fig. 2. The patch stands out distinctly, and is sharply cut off from the surrounding, healthy placenta. In the very earliest stage this is all that is seen. When less recent, however, it is edged all round by a kind of capsule, seen on section as a thin whitish or yellowish streak (Plate I., Fig. 1). Throughout the patch, black points and rounded spots are often visible, corresponding to blood in engorged villous vessels. In this stage the spongy character, though partly lost, has not quite gone.

(2) In this stage the colour becomes lighter. The dark red or purple becomes dusky brown, chocolate-coloured, or brick-red. The spongy character of normal placental tissue has disappeared, and the patch cuts solid, the section showing a smooth surface all over (Plate II., Fig. 1).

(3) In the older infarcts the colour becomes progressively lighter. The dark brown of the preceding stage becomes light brown, yellow, and then sometimes almost pure white.

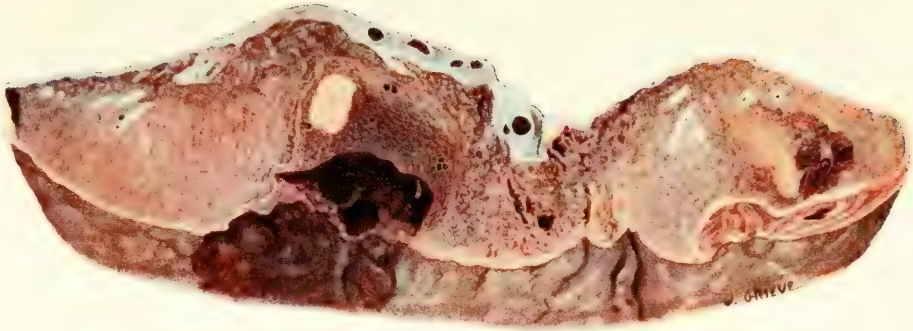
That such a description of infarcts is the true one is proved by the fact that one can find, often in the same placenta, all the various stages between the earliest, purple, and the latest, white, stages. In Plate I., Fig. 1, is shown a large infarct surmounting

Fig. 1.—Placenta from Case of Albuminuria. Shows recent retro-placental clot subtended accurately by a recent infarction, which is marked off from the surrounding placenta by its darker colour and solid appearance. There is a suggestion of a "capsule." This really means a commencing disappearance of the hæmoglobin in the blood-cells, a change which, in one part, has occurred to such an extent as to produce a pale infarct.

Fig. 2.—Placenta in Case of Eclampsia. In one part a large purple or recent infarct is seen, with no naked-eye indication of how it has been formed. At another region a retro-placental clot is seen surmounted by an older infarct: the colour is becoming paler, the solution of hæmoglobin being most marked at the periphery. This gives the appearance of a "capsule." This fig., therefore, shows two stages in the infarction process. By the time the pale stage, and perhaps even the purple stage are reached, the toxic products of necrosis have diffused into the blood circulating freely round about in the healthy placenta.

PLATE I.

PLATE I



a large clot. That it is not quite recent is shown by the yellow "capsule," and by the fact that one part has passed into the pale stage. No doubt if it had been left the whole infarct would soon have become similar in appearance.

What is the explanation of these changing colours? Why is the later stage pale whilst the early is dark? The first explanation that suggests itself is that it is associated with an alteration in the blood contained within the affected portion. That such is the case is proved by a microscopic examination of the various phases. To begin with, the villi are engorged throughout, the thin-walled capillaries in the small villi and the thicker vessels in the large villi are greatly expanded and are tightly packed with blood cells. In the small villi the vessel expansion may be so extreme that the stroma has become displaced to the periphery, and there intervenes between the blood in the villi and that in the intervillous space only a very thin sheet of tissue (Figs. 2 and 3). The purple colour of the fresh infarct is therefore due to the villi being turgid with poorly oxygenated blood. The whole appearance in this stage closely simulates the vascular hyperæmia and stagnation which occurs in other tissues when there is any local lowering in vitality, such as might result, for example, from injury. To my mind the engorgement which is present in the early infarction is of a smaller origin. It is an evidence of local reaction which precedes the local death, that is inevitable when the blood supply of the part is cut off. In the later stages the blood within the villi, and any blood present in the intervillous regions, becomes paler and paler. The blood cells absorb the staining matter less and less. The hæmoglobin becomes completely removed and nothing but the shadowy outlines of the corpuscles are seen; or there may be a crumbling process, which results in a kind of granular debris—all that is left of the original blood cells. These stages correspond to the later stages of the infarction, and

demonstrate readily the meaning of the phases presented by the changing nodule. It would thus seem that one of the chemical alterations occurring in the blood pent up in the vessels consists in a change in or solution of the hæmoglobin. Müller has shown that a similar change occurs during autolysis in the lung, and Mathes has reproduced the same change in the placenta by autolysis. In my experiments on placental autolysis, which I will record later, I have noticed the same result.

In association with these changes in the vessels and stroma there are other important structural alterations in the infarcted placenta. These have been carefully described by other investigators. One of the most frequent is a somewhat remarkable one. It consists of a *close packing of the villi* together throughout the whole or part of the infarct. In many cases the spaces between the villi are occupied, sometimes tightly, with blood cells. There may be at the same time a deposit of fibrin. This blood undergoes disintegrating changes similar to those already referred to in the villous blood. In the condition, however, which we are considering, the intervillous space is empty or nearly so, and the villi, instead of being separated from one another by comparatively wide intervals, as in the normal regions round about, have come close together. They may simply lie opposed to one another, or there may be an actual cohesion or fusion of the epithelial surfaces (Figs. 2, 3, and 4). In such cases the vessels are usually, as in other infarcts, greatly expanded, but not necessarily so, and the condition cannot be attributed to a general increase in the size of the villi, caused in this way, with a corresponding crowding of them together. Eden, so far as I know, first specially pointed out this rather remarkable condition, and explained it as due to a "progressive diminution of blood supply to a part of a cotyledon, by obliteration of a maternal artery [which] would cause the villi to become

Fig. 1.—Placenta from Case of Severe Albuminuria. Hæmorrhages partly retro- and partly intra-placental, surrounded by areas of necrosis imbedded in and surrounded by healthy placenta. Note how accurately the infarcts are related to the clots, an appearance which indicates that the necrosis is due to the interference with the maternal blood supply consequent on the hæmorrhage. The infarcts are fairly recent, corresponding to the intermediate stage. Towards the periphery of one infarct, however, the colour is becoming paler, due to the solution of the hæmoglobin. On one side of the central clot the placenta is healthy, the nourishment here being maintained.

Fig. 2.—Placenta from Case of Acute Toxæmia. Large retro-placental clot (accidental hæmorrhage) is seen, subtended accurately by a deep red or recent infarct. This is clearly formed as the result of the interference with the maternal blood supply in this part. To one side a smaller old infarct, and further along an infarct of recent formation, though beginning to become pale, are seen.

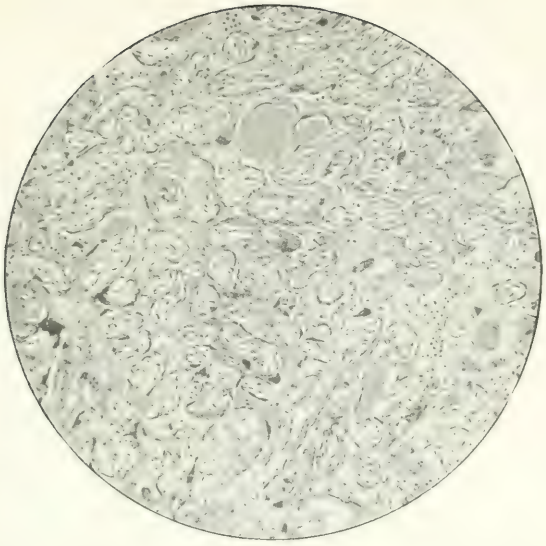


Fig. 3.—Old (white) infarct. Villi packed together. They are degenerated and poorly staining. The expanded vessels are well seen. The blood cells in these cases are pale, due to a disappearance of the hæmoglobin. Case of Albuminuria.

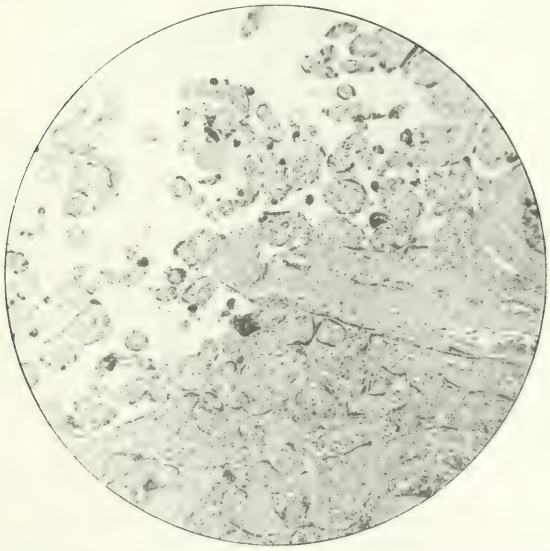


Fig. 4.—Edge of recent (red) infarct. To the right the densely packed villi are seen, to the left the healthy placenta. Case of Albuminuria.

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crowded together because there would not be sufficient blood in the part to maintain them at their normal distance from one another." The pressure exerted by the rest of the placenta "would drive the villi together into a closely crowded, consolidated mass." This I believe to be the real explanation of the process. In other words, it must be looked upon as one of the possible changes where the local blood supply is disturbed. It will occur where the venous channels remain patent, or where the surrounding placental pressure can squeeze out the blood after obliteration of the afferent supply has taken place.

Clotting in the Intervillous Blood Space.—As I have already stated, one never finds any trace of coagulation in the intervillous space in the healthy placenta. In infarction a noteworthy appearance is the formation of fibrin, in the meshes of which red blood corpuscles are often entangled. *That this fibrinous deposit is not necessarily secondary to the changes in the villi is shown by the fact that we can often find it in conditions in which we definitely know there has been a stagnation of the maternal blood, and where the villi are healthy*—e.g., *accidental hæmorrhage.* As I shall show later, the coagulation is primary; the necrosis, which later on occurs in such cases, being secondary. As the result of the examination of a large number of placentæ, healthy and diseased, I have convinced myself of the truth of this statement. When we find a fibrinous deposit between the villi, we can be sure that there is some grave interference with the maternal circulation in the corresponding part.

The *other changes* in the infarcted regions, which result in a gradual disintegration and softening, have been fully referred to by other writers, and need not be detailed here. The epithelial layers soon show changes. In the earliest stages a proliferation of the syncytial nuclei, forming masses of darkly staining tissue, would seem to be a sign found specially

and characteristically in this condition. Later on both layers of the epithelium become pale and eventually crumble away. Similar changes attack the stroma and the cells of the vessel walls. In the later stages the autolytic process results in a solution of the tissues, with the result that cavities may be formed in the centre of the infarcts. The degenerative changes often seem to start and progress most rapidly towards the centre of the nodules. In other cases they commence at the surface, as in the left-hand side of the infarct in Plate II., Fig. 1, or in an eccentric position, as is shown in Plate I., Fig. 1.

III. *Factors responsible for Infarction.*

Whilst an understanding of the exact origin of localised placental death is not essential to my interpretation of the toxic process, I believe that the ultimate explanation of albuminuria and eclampsia must remain hidden until light is thrown on these changes. There have, in the past, been two opposing ideas regarding the origin of placental infarction:—

(1) Eden, Whitridge Williams, and others believe that it is due to a change commencing primarily in the villi themselves. Their blood supply is interfered with as the result of an obliterating change in the vessels. This leads to a degeneration of the epithelium and stroma of the villi, and secondarily to a coagulation of the maternal blood.

(2) Other investigators, such as Hofmeier, believe that the degeneration is due to an interference in some way with the maternal blood supply.

The first, and, in this country, the most commonly accepted idea naturally assumes that the villi are dependent for their nourishment upon the fœtal blood supply, and that once this is obstructed they must undergo progressive necrosis. If one could show definitely that the villous structures are independent of the fœtal blood, and, moreover, can live and proliferate when

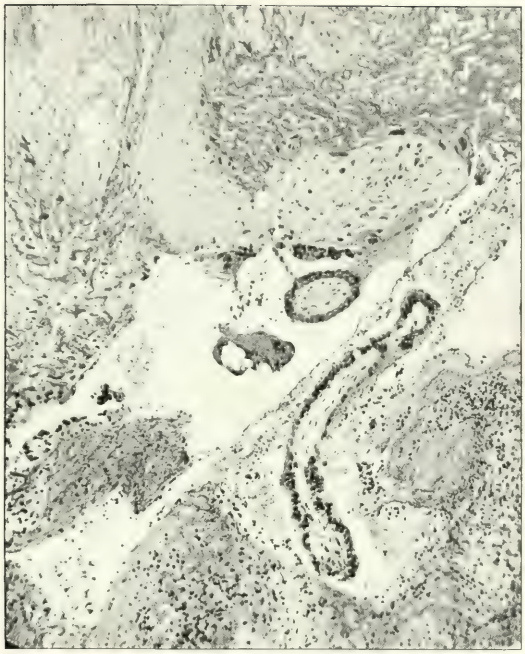

Fig. 5.—Tubal Pregnancy. Above, the necrotic villous trunks are seen strangled in blood-clot. Below, the tips of the same villi are seen to be healthy where they lie in spaces in which the maternal circulation is maintained.

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this is removed, so long as the maternal supply remains intact, it would render this explanation of the infarction process untenable. This is not difficult to do. There are several considerations which show it:—

(1) The time when the chorionic elements are most active and proliferate most rapidly is during the early stages of the development of the ovum, when there are, as yet, no fœtal vessels formed, and where the trophoblast and its villi obviously live directly upon the mother's blood.

(2) In hydatid mole the chorionic villi live, and, as we know, actively proliferate, when there is not a trace of a fœtal vessel, and when the entire nourishment is derived from the blood of the mother. The same is true of chorion epithelioma.

(3) In tubal pregnancy one can sometimes recognise the independence of the villi of the fœtal blood in a diagrammatic manner. Where there has been a considerable hæmorrhage into the extra-chorionic space great masses of villi become strangled in blood-clot. I have seen one such case where all the trunks in the neighbourhood of the chorion had undergone fibrinous necrosis, but near the tube wall, where, in parts, the maternal circulation was unimpared, the tips of the necrotic villi remained healthy (Fig. 5).

These facts demonstrate beyond doubt that the villi, even after the fœtal blood supply is removed, can live, so long as the maternal supply remains uninvolved.

That the localised patches of dead tissue in the placenta are dependent upon an interference with the maternal blood supply is indicated by other findings of a positive nature:—

(1) As I have pointed out, in by far the largest number of cases, the infarcted areas lie in relation to the decidual surface. Moreover, in every such case one can detect degeneration, often necrosis, of the decidua. It is obvious that were infarction due to maternal causes, this is the disposition that would obtain.

(2) In large infarcts, resting upon the decidua, one some-

times can see, even with the naked eye, small nodules of healthy placenta on the outer or decidual aspect, which are obviously cut off entirely from the main part of the healthy placenta. Tracing in sections cut by the knife is sufficient to show this in such cases. This complete isolation of a healthy patch in a dead mass of tissue can mean one thing only, that whereas the remainder of the infarcted portion has lost its nourishment, the healthy nodule has retained its maternal supply. It is obvious that such a finding must be one of rare occurrence, but I have seen it twice, and in each case the healthy piece has rested against the decidua, and into it healthy maternal vessels could be seen opening.

(3) *Retroplacental or Intraplacental Hæmorrhage as a Cause of Infarction.*—A consideration of the changes associated with placental hæmorrhage provides one of the most convincing proofs of the maternal origin of infarcts. Where there has been a hæmorrhage in the decidua serotina or into the placenta itself, there will be apt to be a local deprivation of the corresponding part of the placenta. The discovery of necrotic changes in these regions would be strong confirmatory evidence of the thesis I am trying to prove. As a matter of fact, it was the discovery of this evidence that first convinced me of the fallacy of the usual explanation. One of the most surprising features of this investigation has been the great ease with which such evidence, and that of an unequivocal nature, has been obtained when it was looked for.

Where a retroplacental hæmorrhage is not quite recent one invariably finds the adjoining placenta diseased. The extent to which the necrotic process has gone depends upon the time during which the circulation has been cut off. Over old clots we see old, yellow, or white infarcts, whereas over young clots the infarction process is only in an early stage. Over clots of recent formation it may only be recognised on careful examination with the unaided eye, or it may not be evident without

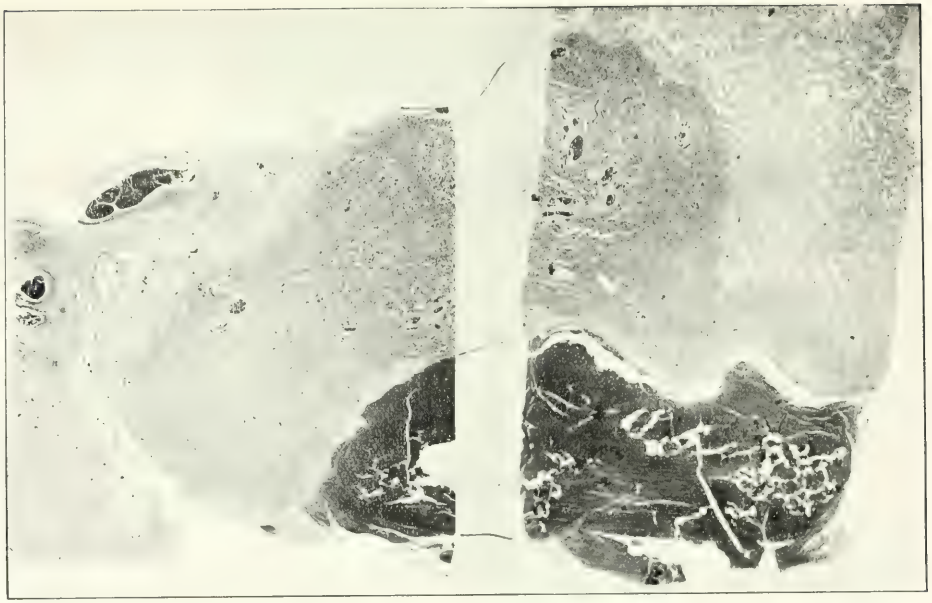

Fig. 6.—Recent (red) infarct accurately subtending retroplacental hæmorrhage. Shows dense packing of villi. The naked-eye appearance of this infarct is shown in Plate I., Fig. 1. Case of Albuminuria.

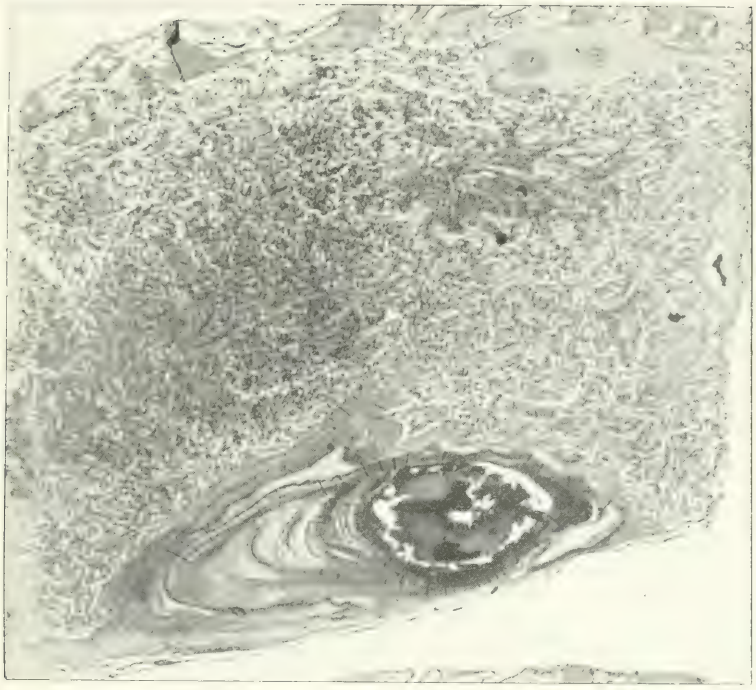

Fig. 7.—Recent infarct (invisible to naked eye in fresh placenta). The early stage of the consolidation process is seen in a region where the blood supply is cut off by thrombosis in the underlying vessel. Case of Eclampsia.

microscopic examination. In the earliest stages recognisable to the naked eye it consists of a deeply stained purple or dark brown patch, often difficult to recognise until the blood has been allowed to diffuse from the adjacent healthy placenta. In Plate I., Fig. 1, this appearance is shown. The necrotic area was at first difficult to recognise. It is seen to be not quite recent, because it is sharply outlined by a pale yellowish margin. Figs. 6, 7, and 9 show infarcts subtending retroplacental clots (see also Plate II., Figs. 1 and 2).

In view of what I have said there can be little doubt that we are here brought face to face with an infarct in the making. The objection that might be urged, that the hæmorrhage is secondary to the necrosis, is disposed of by the appearance, a common one, seen in Plate II., Fig. 1. Here the solid infarcted portions are seen closely and accurately nestling round the clot. If the necrosis had been primarily developed it is obvious that any bleeding into the placenta would not have chosen to so displace the solidified tissue. It would have found much less resistance in the loose retroplacental decidua.

As is well known, hæmorrhage in the placenta is commonly associated with toxæmic states. Not infrequently, in fact almost always, one finds masses of blood-clot of varying age adherent to the maternal surface. The possible ætiological importance of these clots, to which formerly little attention has been paid, will be evident from the statements just made, and this ætiological significance will be seen to force itself upon us even more clearly when we study the conditions that obtain in accidental hæmorrhage. The bleeding, in the cases we are dealing with, is most frequently retroplacental. Over the blood there is a thin layer of compact decidua, which is invariably necrotic, when the condition has been present for a sufficient time. In other cases the bleeding may be within the substance of the placenta. Here again such clots are often surrounded by a necrotic layer of placenta (Plate II., Fig. 1).

Whilst the observations I have just recorded are amenable to one interpretation only and supply us with evidence proving that, in these cases, the necrotic placenta is dependent upon an involved maternal circulation, in many cases, in fact the majority, we do not find this unmistakable evidence. It is true that the majority of infarcts are related to the decidual surface, but in many it is impossible on naked-eye or microscopic examination to be sure of their exact mode of origin. The decidua is usually diseased and the vessels are thrombosed, but these changes might quite well be, considered in themselves, secondary, and have usually been so considered. A little thought, however, will show that a very likely explanation of the hæmorrhage in the decidua, to which I have called attention, would be blockage of the decidual veins or the veins in the muscular wall—*e.g.*, by thrombosis. This, in the presence of a free arterial supply, would tend to a backward pressure in the thin venous channels and would readily account for a hæmorrhagic escape, situated as they are in a soft unsupported decidua. Such an explanation has been actually advanced by Veit as the cause of the retroplacental bleeding in accidental hæmorrhage, though he has attributed it to a blockage of the veins by masses of deported villi, and not to thrombosis. Be this as it may, I believe that the almost certain factor operating in these cases is a blockage in the veins, and the frequency with which thrombosis is found in the decidual vessels and veins of the muscular coat suggests rather that the responsible agent is an excessive occurrence of this change, which is so frequent during the later months that it may almost be considered normal (Friedländer). Where the vein also is blocked hæmorrhage will be the result. But this will only be one way in which the vascular changes will manifest themselves. *If there is a blockage of the artery, anæmia of the corresponding part of the placenta will occur* and this will be the only sign of the vascular involvement. That such is a very

common factor in infarct formation is shown by the fact that a majority of the infarcts in my specimens show a close packing together of the villi into solid masses containing no blood in the intervillous space, *as if a kind of collapse has occurred subsequent to a cutting off of the entering blood*, just as collapse of the lung follows obstruction of a bronchus (Figs. 6, 7, 8, and 9).

We may now summarise in a few words our conclusions regarding the origin of infarcts. In the first place, there can be no doubt that the chorionic elements are dependent immediately and directly upon the maternal blood for their nourishment, and can live and flourish where the fœtal blood is absent. This fact renders untenable the belief that infarction is due to an obliterating change in the vessels of the villi. A recognition of this fact leads inevitably to the conclusion that necrosis in the placenta will occur if the maternal blood supply is interfered with. I have shown that we can often find direct and undoubted evidence of such necrosis occurring in regions where there has been an arrest in the corresponding maternal supply of blood. Where there is an obstruction in the vein or veins hæmorrhage will be more likely to occur and will act as a signal post indicating the presence of the obstruction. Where the artery or arteries are involved, alone or in conjunction with the veins, anæmia of the part of the placenta concerned will occur, followed again by necrosis. In such cases it may be difficult to find absolute anatomical proof of this mode of origin, but we may glean hints of its occurrence by a careful examination of the changes.

Remarks on the Maternal Circulation in the Placenta.

The observations I have just recorded indicate the fallacy of the usual idea regarding the circulation in the placenta. This is usually considered to be so free throughout, that any

interference in one part is quickly made good by an inflow from the adjacent regions. The placenta is not to be looked upon as a loose sponge in which there is a kind of irregular mingling of the maternal blood. So far is this from the truth that I believe it more likely that, just as in other tissues, each portion is supplied by certain maternal vessels and relies on them for the nourishment. By injecting some colouring matter into the centre of an isolated lobule one can easily satisfy oneself of its isolation from the adjacent placenta, so far as its blood supply is concerned, for only with great force can we drive the fluid beyond it into the other parts. We can easily understand that this must be so, when we remember that, in the natural state, the fissures between the cotyledons are occupied by solid columns or partitions of decidua, which often run up throughout the entire thickness of the placenta. During separation these septa are left attached to the uterine wall. The separation is rendered easy in these regions by the œdematous loosening of the decidua immediately under the compact layer, which occurs in the later weeks of pregnancy, and to which I have already alluded. Not only are the cotyledons independent of one another so far as their vascular supply is concerned, but differing parts of the same cotyledon are dependent on different vessels for their nourishment. In each of these areas there is a complete circulation, though, at the boundaries of such circulatory systems, it is probable that there is a mingling of the bloods.

Relation between Accidental Hæmorrhage and the Toxæmias.

Accidental hæmorrhage is a subject of great importance in connection with the thesis I am attempting to establish. The evidence which it provides is pertinent to the discussion and is even more convincing and less confusing than that to which

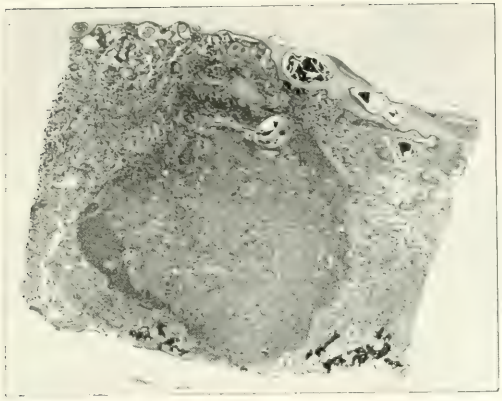

Fig. 8.—Recent (red) infarct. Shows consolidation process well advanced. In this there is no sign indicating how the maternal blood supply has been cut off. This information is often absent. Case of Eclampsia.

Fig. 9.—Recent (red) infarct subtending retroplacental clot. This infarct was invisible to the naked eye. Case of severe accidental Hæmorrhage without Albuminuria.

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attention has been directed on the preceding pages. In accidental hæmorrhage we have a retroplacental bleeding, which occurs with greater or less rapidity and leads to a corresponding amount of placental detachment. It must be considered as merely an exaggeration of the hæmorrhage in a similar site, which is fairly common in the last weeks of pregnancy. There are all grades between the smaller hæmorrhages (Plate I., Fig. 1), through the comparatively large bleeding shown in Plate II., Fig. 2, up to the condition we are considering, where there is an excessive loss of blood. The special interest which this condition possesses for us resides in its close relationship to the toxæmias, albuminuria and eclampsia. Its affinity with albuminuria has been recognised since it was pointed out many years ago by Winter. Its relation to eclampsia was referred to by Essen-Möller in a contribution to the recent International Congress in London. Essen-Möller reported two cases from his own clinic which were associated with eclampsia. Seitz has recorded two, Winter one, Harry one, and Hartmann two. Moreover, Bar and Kervily have found in one case, where there were no fits, degenerative lesions in the liver similar to those present in eclampsia. (I quote these references to the literature from Berggren's paper.) Whilst this is so, one thing emerges clearly from the investigations on this relationship and is expressed by several of the authors—namely, that *it is impossible to look upon the toxæmia as the cause of the bleeding. For, in a large number of the cases, there is no trace of a toxic state.* For instance, in eight cases collated by Berggren, albuminuria was present only in four; in Zweifel's twenty-one cases there were thirteen cases without albuminuria. Essen-Möller brings out this same fact in a striking way. In 7000 accouchements at the University of Lund, albuminuria was observed in 11 per cent., whilst accidental hæmorrhage was observed in only 0·45 per cent. He therefore says that "if albuminuria is a cause of retroplacental hæmorrhage, it is a

cause of no great activity. But it is equally necessary to consider it from an opposite point of view. Out of my twenty-nine cases eleven showed albuminuria, and this figure rises to seventeen if we include cases with a trace only. So that whilst albuminuria is seen in only 11 per cent. of confinements, it is seen in 37 to 50 per cent. of cases of accidental hæmorrhage. It therefore seems to me that we can draw from these facts a second conclusion—namely, that the frequency of albuminuria in retroplacental hæmorrhage suggests something more than the idea of a coincidence pure and simple."

Such a condition of affairs, otherwise an admitted mystery, is readily explained in terms of the interpretation of the toxic states which I have already advanced. In accidental hæmorrhage we obviously have a corresponding part of the placenta cut off from its blood supply. If the hæmorrhage develops and extends so rapidly that it quickly kills the patient or determines a complete separation of the placenta, either naturally or by the interference necessitated in the patient's interests, there will be no opportunity for the necrotic changes to develop and there will be no toxæmia. The nature of the condition is such that termination in one or other of these ways must be common, and readily accounts for the fact that in about 50 per cent. of such cases there is no toxæmia. But, if the placenta remains attached at one part for some hours or days, the circulation there will remain undisturbed, and there will be an opportunity for the discharging into the maternal blood of the toxic ingredients, quickly elaborated by the disintegration of the separated portion. Only in such cases will an albuminuria or an eclampsia develop. As in the retroplacental hæmorrhages of smaller amount, so here it is easy, in cases where the blood-clot is not quite recent, to demonstrate the infarction changes in the severed portion or portions. The degree to which the change is manifest depends on the time that has elapsed since the bleeding occurred. I have had an opportunity in my specimens

of recognising only the earliest stages. When present they are, however, distinct. On some occasions they have been found only after microscopic examination in a placenta that looked quite healthy to the naked eye. The discovery of these changes must be looked upon as strong confirmatory evidence in support of the truth of the reasoning which had foretold their existence. I shall refer, later on, to the question of the ætiology of accidental hæmorrhage, in so far as we have facts that permit of such an investigation.

I regret that a complete clinical history, so far as albuminuria is concerned, was obtained in none of my specimens. Whilst this would have given added interest to the discussion, it is, however, not essential. For the study of the anatomical changes in accidental hæmorrhage I have had one complete uterus, to which the placenta is still attached, and eight placentæ, as well as pieces removed from the uterine wall from four cases of fatal accidental hæmorrhage.

Changes in the Placenta.

The alteration in the placentæ varies with the age of the clot and the amount of separation that has occurred. In one case, in which there was not much hæmorrhage, there was no albuminuria. The labour ended soon after the first onset of the bleeding. In it an interesting appearance was found. The blood-clot was quite fresh. The placenta was examined soon after removal, and it was noted that the part of the placenta (about a quarter of the whole) that had been severed from the uterine wall was deeply congested as seen from the amniotic surface, the remainder being quite pale. This appearance corresponds to the earliest stage of the degenerative process, which is characterised by an expansion of the vessels in the villi. It would seem to be analogous to the congestion that occurs round an infarct in other parts of the body. The unaffected vessels undergo a rapid engorgement after the cutting

off of the blood to the affected part. In the placenta the vessels of the villi, in which the circulation is primarily uninvolved, undergo a similar engorgement. For the rest, the degenerative changes in the placenta are similar to those which I have already described under the heading of infarction in general. In this condition, of course, they are more extensive, as large masses of the placenta, or even the whole placenta, are apt to be deprived of their blood supply. The changes may be equally developed in the affected parts, or, where differing regions have been detached at different times, the changes may be more marked in some places than in others. One of the first changes to develop is a *fibrinous deposit in the intervillous space.* As I have already pointed out, this is to be looked upon as a certain sign of severe vascular involvement. It may be present before any visible changes have occurred in the chorionic elements. In the majority of cases, however, where it is found there has occurred a congestion of the villous vessels (Fig. 2).

In Fig. 9 is seen another typical appearance found in these cases. A small recent clot is adherent to the decidual surface. Subtending this clot there is a region of definite consolidation in the placenta ; in other words, a commencing infarct. It is localised to the region of the clot, and, even on naked-eye examination of the section, *the villi can be seen to be densely packed together,* the looser placental tissue all round being seen. Under higher magnification fibrinous threads are seen between the villi, and the vessels in the villi are greatly expanded. The epithelial covering of the villi is not obviously diseased. This section is taken from a placenta near a huge clot which had severed practically half of its surface from the uterine wall. As the result of the accumulating blood the placenta had been so compressed that its thickness was reduced by one-half, and in some places by two-thirds. Throughout this enormous piece of placenta changes similar to those just described were found —*i.e.*, there had taken place a huge early infarction.

At first sight it might seem possible to explain the degeneration in these cases as due to the compression of the blood-clot, and no doubt this must play a part in the devitalising process. That it is not the necessary factor, however, is shown conclusively by the occurrence of the changes in placentæ, in which a ready detachment has occurred without any obvious compression having taken place. This is, as a matter of fact, the usual condition of affairs. In these cases we find the intervillous coagulation and the congestion of the villi in the earlier stages, and the necrotic changes in the villi in the later stages. As I have said, a packing of the villi into solid masses, suggesting a collapse of the placental substance, is present in the majority of such cases. It is doubtless due to the anæmia of the part attendant on the interference with the blood entering, and, although the veins are almost certainly involved, and a leakage away along the normal channels is thus rendered impossible, the forcing of the blood out of the intervillous spaces into the surrounding, less involved, intervillous regions is probably a matter of comparative ease. Especially is this likely in view of the fact that at the confines of the individual circulatory systems in the placenta there is probably under ordinary circumstances a certain mingling of the bloods. *In any case the appearances leave no doubt of the fact that when the blood supply of any part of the placenta is interfered with, collapse and solidification is likely to occur.*

The appearances in the one case of accidental hæmorrhage in which I have been able to make a complete study of the placenta *in situ* are interesting. The placenta was completely separated by a huge blood-clot except at one edge. The hæmorrhage is completely retroplacental, a fine layer of compact decidua covering the outer surface of the placenta. There were, throughout the separated part, changes such as I have referred to. The placenta is pale throughout, except at its attached edge, where the most recent disintegrative change is

seen, in the shape of a large purple patch. It is clear that this is just the region where the most recent evidence of a degenerative process will be evident. It is the last part to be separated. As I have indicated on a preceding page, the purple colour soon gives place to a paler hue as the autolytic process leads to a solution of the hæmoglobin. This is the condition of the remaining part of the placenta. After an examination of such a specimen one can be left in no doubt whatever that the disease in the placenta is secondary to the deprivation of the maternal blood supply.

Changes in the Uterine Muscle, Decidua, and Vessels in Accidental Hæmorrhage.

The chief subject for this study has been the complete uterus, to which I have just referred. For this specimen I am indebted to Professor Kynoch, of Dundee. I have, in addition, been able to study sections cut from four complete uteri from fatal cases of accidental hæmorrhage. These are in the Museum of the University of Liverpool, and, naturally, the chance of anything like a complete investigation of them was impossible. For them I am indebted to Professor Briggs. My remarks will be chiefly directed to the specimen of Professor Kynoch. In it a thorough examination was made. The other specimens served to provide confirmatory evidence of much that it demonstrated. The complete specimen consists of the uterus removed after death. The specimen was shown to the Edinburgh Obstetrical Society last year by Professor Kynoch, and I need not refer to the clinical history. The uterus is occupied by an immense blood-clot. The placenta has been detached by this, except along one edge, where it is still adherent to the uterus. There are marked and important changes in the muscular wall and in the decidua.

Changes in the Decidua.—These were discovered purely by

Fig. 10. Accidental hæmorrhage (Professor Kynoch's case). Piece of uterine wall beyond the placenta. To the left the amnion and chorion are seen, then the decidua vera showing greatly expanded vessels and hæmorrhage. To the right the muscular wall beset with small hæmorrhages. (It is therefore a case of "diffuse utero-placental Apoplexy.")

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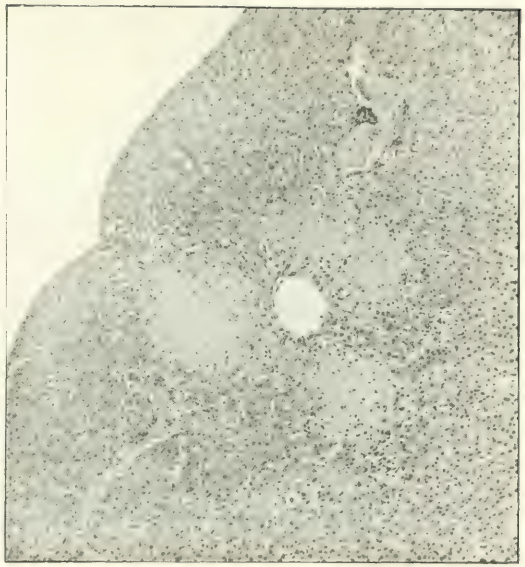

Fig. 11.—Liver of guinea-pig injected subcutaneously with extract of autolysed human placenta. Shows "focal necrosis" similar to that found in the human liver in eclampsia.

accident as the result of the way in which the specimen was cut. The decidua vera is deeply congested. In the sections the *vera* is marked by a deep red line, and is obviously the seat of diffuse hæmorrhage or of vascular congestion. Microscopically the appearance was found to be due almost entirely to an enormous expansion of the decidual vessels into thin-walled sinuses. Here and there areas of hæmorrhage are present (Fig. 10). In the adjoining muscular wall the vessels are in a state of congestion, and surrounding them there may be scattered hæmorrhages, but the changes here are nothing like so evident as in the decidua. The congested vera can be traced directly (even by the naked eye) into the serotina under the placenta, where the same vascular changes are present in an even more marked degree. In the *serotina* the vascular expansion is excessive, and there is an extensive blood leakage into the surrounding tissues. It is obvious that it is to the congestion and hæmorrhage here present that the enormous blood escape into the retro-placental space is due.

Changes in the Muscular Coat.—In no place is there any evidence of degeneration of the muscular tissue. In many places there is an expansion of the vessels, and an œdematous escape. In the muscular coat underlying the placental site (and also, but to a less extent, in that under the congested vera) the vascular changes are most evident, and here a hæmorrhagic leakage through the vessel walls has occurred. These appearances Essen-Möller and others have previously described. In one of my other specimens there was an extensive hæmorrhage into the muscular coat, which was visible as a deep purple patch under the peritoneum over the greater part of the anterior surface of the uterus.

It is evident, from a study of these changes, that the vascular disturbances present in accidental hæmorrhage are widespread, and in any attempt to account for them one

must recognise that they are found throughout the uterus, and are not confined to the placental region. It has been amply proven that a toxæmic state cannot be considered as an ætiological factor, for in 50 per cent., if not more, there is no albuminuria. In discussing the causation of the milder degrees of retroplacental bleeding it was pointed out that a blockage in the veins of the decidua or adjacent muscle might quite well explain the condition. As a matter of fact, this explanation has been advanced by Veit to account for accidental hæmorrhage, his belief being that a mass of deported villi may plug the vein. In my belief this is an unlikely explanation, for it is difficult to see how a mass of villi sufficiently large could be severed from the placenta, though from Veit's sections which I have had the opportunity of examining, it is evident that there may occur a very luxuriant growth of villi along the veins adjacent to the placenta. To my mind a thrombosis in the veins is a more likely explanation. It is well known that thrombosis of the veins of the decidua and uterine muscle is so common that it may be looked upon as a normal change in the later weeks. It is also known that during pregnancy there is a very special tendency to the excessive formation of thrombi in the veins throughout the body. A blockage of the small veins near the placenta may quite well account for the smaller degrees of hæmorrhage in the retroplacental site. For the vascular changes present throughout the uterus in cases of accidental hæmorrhage are so widespread that it is evident that, if blockage of the veins is the cause, it must be a blockage that is far back in one of the main venous trunks in the uterus or even in the broad ligament.

As I have said, I had only one specimen in which a complete examination of the changes was possible. In it I decided to investigate the main ovarian and uterine branches, and, to my surprise, without the least difficulty a massive,

extensive, and fairly old-standing thrombosis was found in the ovarian vessels on each side, especially in the left. The uterine vessels seemed to be healthy. *It is therefore probable that accidental hæmorrhage is due to a thrombosis in a main venous trunk in the uterine wall, or even in the pelvis. The thrombosis probably occurs slowly, and may be present without any untoward signs, so long as it is not sufficient to obstruct the lumen of the vein completely. Let the obstruction become complete, especially let this occur suddenly, and the great venous pressure thrown back throughout the affected regions of the uterus will at once lead to the changes described.*

The main purpose of this investigation was not a discussion of the ætiology of placental infarction, or even of the ætiology of accidental hæmorrhage. I have devoted considerable space to their consideration, because I believe that the usual interpretation which is placed upon them has blocked the way for an enlightened effort to fathom the exact relationship existing between the toxæmic states (albuminuria and eclampsia) and placental disease. Hitherto this relationship has been considered as, in many respects, a mystery. This attitude, as I have tried to show, was due solely to the failure to recognise that placental disease must be considered as due to a cutting off of the maternal blood supply. *Whatever be the cause of the vascular interference, it is certainly not due to a toxæmic state. The study of the anatomy of placental infarction showed this, and the investigation of the conditions present in accidental hæmorrhage have proved it. It is clear, then, that if placental disease is especially prone to exhibit itself in association with a toxæmic state, and if it can be shown to be neither due to this toxæmic state nor to any condition accompanying this toxæmic state, the only solution of the riddle is that the necrotic placenta is the source of the poison.*

There is no method of evading this conclusion. I pointed out early in this research that the clinical evidence in ordinary

cases of albuminuria and eclampsia, taken in conjunction with the anatomical, strongly suggested that this was the true explanation of the relationship. An exactly parallel study carried out in regard to accidental hæmorrhage leads to the same conclusion.

Experimental Reproduction of Eclampsia in Lower Animals.

The anatomical investigations recorded in the preceding pages indicated that the poison or poisons responsible for albuminuria and eclampsia are elaborated in a dying placenta. They, moreover, led to so precise a knowledge of the exact manner in which this process occurred that it seemed likely that an imitation of it *in vitro* should enable us to isolate the toxic material for experimental purposes.

One fact issued clearly from these investigations—namely, that the poison must be elaborated early in the course of the disintegrative change of the placenta; for there may be a fulminant toxæmia with an apparently healthy placenta. In severe toxæmias, however, if several days elapse before the birth of the placenta, massive, recently necrotic areas are visible. The time during which the placenta has been retained has allowed of the evolution of the devitalised patches into visible, dark red, or purple nodules of solidified tissue. In my earlier experiments this fact was not grasped. The placenta was autolysed for eight days or longer, and, although a suggestion of success was every now and then obtained, the results were, on the whole, disappointing. With a clearer understanding of the exact process the results were positive all along the line. To begin with, a definite standard, by which the results were to be gauged, was formulated. Such a standard was not difficult to obtain, as the clinical and morbid picture in eclampsia is so precise. An attempt was to be made

to reproduce this in lower animals in its totality. The main objects to be aimed at were: (1) The production of severe convulsions; (2) the reproduction of the liver changes, which, it is well known, are especially characteristic of eclampsia; and (3) the reproduction of the degenerative changes in the kidney.

Convulsions.—This, the main symptom in eclampsia, I had expected, when beginning my experiments, to find developing only some time after the animal had been brought under the influence of the extract of the autolysed placenta; for it has been usually believed that the convulsive seizures are due to the accumulation in the system of some product of faulty metabolism, and are secondary to the liver or kidney involvement. When the proper method of preparing the placenta was discovered, one of the most immediate results of the injection (sometimes developing within twenty or thirty seconds) were severe and prolonged muscular spasms. In many cases the condition was an accurate reproduction of an eclamptic seizure.

Liver Changes.—These are so characteristic, as found in eclampsia, that I decided to make them the chief touchstone by which to test my results. Workers who have specially devoted their attention to these changes almost unanimously testify to their special association with eclampsia. They consist in a diffuse involvement of the organ. On naked-eye examination whitish areas are found scattered irregularly throughout its substance. In most cases small, they may in unusual cases form large white patches. Microscopically, they are seen to consist of "focal necrosis," which, in the case of the younger and smaller patches, are especially distributed towards the periphery of the lobules. In addition, there is thrombosis in the vessels, especially towards the periphery of the lobules, and, in many cases, there are scattered hæmorrhages. So characteristic are these changes that

Konstantinowitsch says that a diagnosis can be made from an examination of the liver alone. The specially characteristic feature is the localisation of the early changes to the outer parts of the lobules. In this respect, also, my results have been positive. The subcutaneous injection of extracts made from autolysed placenta have given liver changes identical in every respect to those just referred to. I have been able to reproduce the focal necrosis, in the earlier stages especially, located in the periphery of the lobules (Figs. 11 and 12). In other cases, large, sprawling masses of necrosis have been found, and, in one animal, necrosis of a great part of one lobe of the liver was produced. Moreover, thrombosis in the vessels at the lobule periphery would seem to be the cause of the necrotic changes. Throughout the liver in these cases a diffuse fatty degeneration of liver cells is often present, as also scattered areas of hæmorrhage.

Kidneys.—The changes in the kidney, though usually found in eclampsia, are neither so consistent nor so characteristic as the liver changes. In many cases they are slight. In marked cases there may be patches of necrosis scattered throughout the organ. The part specially affected is the epithelium of the convoluted tubules, which shows a cloudy swelling, a fatty degeneration, or coagulation necrosis (Schmorl). In my experiments I have been able to reproduce these changes exactly, and it is especially important to note that the special region which the toxic material that I have isolated affects is the epithelium of the convoluted tubules (Fig. 13).

Technique and General Method of Experiments.

The animals used were almost entirely guinea-pigs, and number forty-seven in all. For the extract fresh normal human placentæ were used.

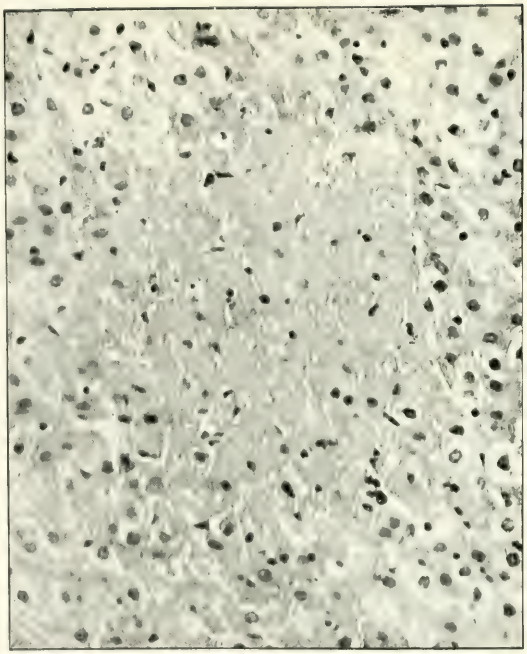

FIG. 12.—Liver of guinea-pig injected subcutaneously with extract of autolysed human placenta. Focal necrotic patch similar to that found in eclampsia.

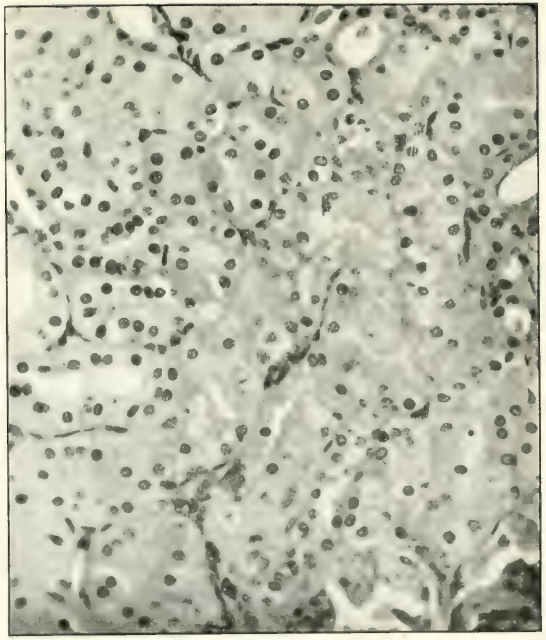

FIG. 13.—Kidney of guinea-pig injected subcutaneously with extract of autolysed human placenta. Convoluted tubules showing evident degenerative changes.

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Method of Preparation of Placenta.

To begin with, this caused considerable anxiety and trouble. As I soon discovered, it is well-nigh impossible to obtain a placenta in an absolutely sterile state. Some method had, therefore, to be discovered of preventing the growth of organisms, whilst allowing the natural ferments of the placenta to act. I first used thymol, adding a small piece of this to the saline solution in which the placenta was immersed. This method I employed when I was allowing the autolysis to continue for seven or eight days. I invariably found that there was a copious development of *Staphylococcus albus* by that time. I next turned my attention to chloroform. This is admirable as an antiseptic, but I suspect that it must inhibit the action of the ferments. In some cases, however, I got a suggestion of positive results with this method. After the autolysis was completed the chloroform was extracted by evaporation *in vacuo*, and the juices were extracted by means of Büchner's press. The methods which I am at present using are three in number:—

(1) Once it was recognised that only a short period of autolysis was required it was thought that, by drying small pieces of placenta in the incubator, the drying process, whilst allowing a short autolysis, would take place so quickly that the risk of infection would be so meagre that it need not be allowed to enter into one's calculations. For this reason small pieces of placenta are washed freely in sterile water, spread out flat on sterile dishes, and placed in the incubator. In twelve or sixteen hours they are quite dry and can be powdered in a sterile mortar. The fibrous structure that remains at the end unpowdered can be easily removed by straining the whole through a fine wire sieve, previously passed through the flame. In this way we obtain a reddish brown, fine powder that can be injected subcutaneously, suspended in saline solution.

(2) As the results obtained with this powder were so strikingly positive, it was decided to employ some other method of autolysing the placenta, which would not be open to even the shred of suspicion, so far as infection is concerned, to which this powder may be held liable. For this reason pieces of placenta, about $\frac{1}{2}$ in. square, were washed free of blood as before, and placed in glycerine and incubated in it. When ready, two or three pieces are placed in a sterile mortar and pounded with 10 c.c. of saline solution.

(3) Recently I have been placing the placenta in saline solution, on the surface of which a film of toluol is placed. The proportions by weight of placenta to saline are two to one. If the whole is thoroughly shaken up before being placed in the incubator, enough of the toluol remains in solution to prevent any organismal growth. This method I began to use when it became apparent that the toxic material or materials liberated by the autolysing placenta were extremely soluble. It was thought that by simply employing the supernatant solution into which the toxic substance had dissolved the same results should be obtainable.

Record of Experiments.

There are several points which require further investigation before a complete publication is possible. For example, I have obtained evidence, during the course of my work, that there are two distinct chemical substances responsible for the eclamptic attack. With the employment of the dry powder marked convulsive seizures are usually obtained, as also the liver changes. With the employment of the glycerine extract, whilst muscular spasms are often present, there are never convulsions, though the liver and kidney changes are obtained in a marked degree. These facts suggest that whilst the powder contains both of the toxic substances, the glycerine

extract contains chiefly that which has a special tendency to produce thrombosis. For these reasons I will record only those aspects of the results which are more or less final in their demonstration, and which are necessary to substantiate the belief that we have isolated the toxic material responsible for eclampsia.

Control Experiments.—A rigorous control was carried out. For this purpose twenty guinea-pigs (which were injected, sometimes over a long period, with extracts of the ovary or corpus luteum) were employed. These experiments were carried on coincidently with mine, and the animals were obtained from the same stock. For the opportunity of examining these control animals I am indebted to Dr A. C. M'Master. In every case the liver and kidneys were examined. In none of them were convulsions, or other immediate signs of poisoning, ever produced. This disposed of the possibility that the spasms obtained in my experiments were merely due to the injection of foreign proteid, etc. In none of the controls were the liver and kidney changes discovered which are characteristic of eclampsia, and which I obtained with the placental injection. I think the absence of the typical liver lesions in such a large number of control animals may be considered as convincing proof of their specificity as found in my experiments.

Details of Experiments.

The injections were invariably made subcutaneously into the loose tissues of the back. By employing this method one had in view the avoidance of a rapidly fatal result, such as is apt to happen with intravenous injection. As other experimenters have shown (Weichardt, Pilz, Freund, etc.), the intravenous introduction of placental extract is quickly followed by death, due to a widespread intravascular thrombosis. Even though the right toxic substance had been isolated in their experiments,

a proper test of its effect, as regards the morbid changes produced, was rendered impossible. Another objection, that of Lichtenstein, was overcome by the subcutaneous method of injection. This worker has shown that the intravenous injection of mineral matter in small particles—*e.g.* sand—may cause results exactly similar to those obtained by Weichardt, Pilz, and Freund, and he believes that these were due, not to any toxic action on the part of the placenta, but merely to the introduction of free cellular elements into the circulation.

Injection of Fresh Placental Extract.—Five animals were injected with the juice expressed from the placenta by means of Büchner's press. This is definitely toxic, if employed in large quantities, doubtless due to the action of a foreign proteid. In two animals 2 c.c., 3 c.c., and 10 c.c. were injected respectively on three successive days. The last dose killed within twelve hours. Three animals were injected with 10 c.c. of this extract at one time and two lived. In none were convulsions produced, and, in those that died, no specific liver or kidney changes were observed.

Extract of Placenta autolysed for Seven to Nine Days in Chloroform.—Thirteen animals were used: 5 to 8 c.c. of the extracted juice were injected once or twice daily till the animals died. In some this occurred after three to four injections, in others not till twelve or fourteen had been given. In one definite and marked convulsions were obtained; in most of the others general muscular spasms. Immediately after the injection the animals always became stupid and dull within a few minutes. In none were the liver and kidney changes produced.

Placental Powder.—It was the use of this that gave the first definitely positive results. Fifteen animals employed: 0·4 or 0·5 grm. of the powder was suspended in 6 or 8 c.c. of saline solution, and injected subcutaneously, usually night and morning. Three or four injections are sufficient to kill. In twelve, convulsive seizures were produced, varying from

extremely marked tonic and clonic spasms of the whole body, lasting from ten minutes to an hour or longer, to general twitchings of the muscles of the head, trunk, and limbs. Other immediate signs of a toxic action were giddiness and a tendency to fall to the side, drowsiness with, in many cases, a complete absence of response on touching the eyes. To anyone acquainted with guinea-pigs these signs will at once assume suggestive proportions. In all, *degenerative lesions in the liver* were obtained; in six, typical focal peripheral necrosis; whilst in two, large, irregular sprawling masses of necrotic liver tissue were found. These appearances are shown in Figs. 11 and 12. Degenerative lesions in the kidney, especially involving the convoluted tubules, were found, but these were neither so evident nor so constant as the liver changes (Fig. 13).

Saline Solution in which Placenta was autolysed for varying Short Periods of Time.—Six animals were used for this. Quantities varying from 5 to 10 c.c. of the supernatant fluid were removed after periods of autolysis varying from one and a half hours to sixty hours. These experiments were primarily carried out for the purpose of determining whether the toxic material passed readily into solution. For this purpose the presence of convulsive spasms was taken as a test. The results in this respect were positive, and showed that even as short a period of autolysis as one and a half hours was sufficient to liberate, at anyrate, the element that provokes convulsions. The spasms are short-lived. Whereas, with the powder, the spasms may last for twenty minutes or longer, with this solution, even in markedly positive cases, the spasms rarely last for more than twenty or thirty seconds. So far, an investigation into the liver and kidney changes in such cases has not been carried out.

Placenta autolysed for Short Periods in Glycerine.—Eight animals were employed. The autolysis was allowed to continue for periods varying from ten to thirty-six hours. It soon

became evident that the extract thus prepared is extremely toxic. Almost immediately after the injection, even of the extract of the short autolysis, the animals become dull and stupid, and often giddy. There may be general muscular spasms, but this is rare, a fact which shows that the specially toxic ingredient must differ from that present in the dried placental powder. Whereas with the powder the animals usually have recovered to a very large extent six or eight hours after the first dose, with this extract they never regain their usual vitality, and remain till the end in a toxic state.

In this connection it is interesting to note that the necrotic changes in the liver and kidney are especially marked with the glycerine extract. They were obtained in all the animals. In one animal the greater part of one lobe of the liver was found necrotic at the post-mortem examination. The presence of well-marked kidney changes is important, in so far as they were not specially developed in the animals injected with placental powder. It is well known that, in eclampsia, the severity of the kidney lesions varies greatly in different cases. Even in fulminant cases they may be slightly marked. These considerations suggest that there are two different toxic agents operating in eclampsia and albuminuria, and that the exact clinical picture, presented in an individual case, depends upon the relative proportions of these two materials present. The one material has a special affinity for the cerebrospinal tissues, and may be called the convulsive agent. The other has a special tendency to produce necrosis in liver and degeneration in kidney, perhaps, as has been suggested by many workers, because of a hæmagglutinative element.

Clinically, it is known that there are two different types of eclampsia. In one the chief symptom is convulsions, though it is usually associated with specially marked liver lesions and often presents kidney changes. In the other, there may be no convulsions. In these cases there would seem to be a special

tendency for severe necrotic changes to occur in the liver and, especially, in the kidney. Under the latter category would come these cases of "symmetrical necrosis of the cortex of the kidneys," which Jardine, Teacher, and Kennedy (of Glasgow), and others have reported. These two different clinical types would correspond to the two different toxic bodies liberated by a dying placenta.

General Summary.

That eclampsia and the albuminuria of pregnancy are due to the liberation of the products of early autolysis of the placenta, has been established by the following considerations:—

(1) The toxæmias are especially associated with recent infarction of the placenta. In severe cases, ending rapidly in labour, there may be no evidence, visible to the naked eye, of placental disease. If, however, the placenta is born several days after the attack, massive necrosis, obviously of recent origin, is seen. It requires some time for the necrosis to evolve into visible form.

(2) Placental infarction is due to an interference with the maternal blood supply of the part. It can be shown conclusively that the chorionic elements are dependent, immediately and directly, upon the maternal blood supply, and, so long as this is retained, can live, even when there is no fœtal supply.

(3) The interference with the blood supply, which is responsible for the infarction, is not dependent upon a toxic state, and, in point of fact, may occur in the most extreme form, where there is no evidence of a toxæmia—*e.g.* accidental hæmorrhage. An examination of the placenta, wherever there is definite evidence of an involvement of the maternal supply, invariably shows disease corresponding exactly to the area of

this involvement. This disease will be evident to the naked eye unless the involvement is quite recent. The study of accidental hæmorrhage was shown to be specially important in this connection.

(4) The placenta is so constructed that, if a part of it die, the products liberated from the dying patch can pass directly into the blood-stream. The organ is unique in this respect. It thus arises that for the occurrence of a toxæmia a circulation of blood round the poison-generating foci is necessary. An understanding of this fact at once dispels many of the difficulties associated with this study. It explains, for example, the cessation of symptoms after the death of the child (and separation of the placenta), and it explains the absence of a toxæmia in cases of accidental hæmorrhage (50 per cent. of the whole) in which the placenta is completely detached by the blood-clot, or by other means. The cases of accidental hæmorrhage associated with a toxæmia are those in which part of the placenta remains attached for some time after the separation of the adjacent part by a retroplacental bleeding. The necrosis of this part liberates the toxic materials.

(5) Where the placental disease is gradual in its onset there is more chance of the evolution of the infarcted patches. This explains why, in long-standing albuminurias, there may be more visible placental disease than in acute eclampsia. It is just the gradual development of the toxæmia that allows of the pregnancy continuing.

(6) These facts all suggest that the toxæmias are due to the autolytic products liberated in the early stages of the placental death. By imitating the process which occurs *in utero*, it has been possible to isolate from the healthy placenta a material or materials of a soluble kind which reproduce the clinical features and morbid changes which all agree are especially characteristic of eclampsia. These are (*a*) convulsions, (*b*) peripheral

focal necrosis in the liver, and (*c*) degenerative lesions in the kidney, especially located in the convoluted tubules.

In conclusion, I would say that, whilst post-partum eclampsia constitutes the only difficulty which I know of in the way of absolute proof of my thesis, I think the evidence I have advanced is sufficiently cogent to necessitate a thorough exploration of the uterus in all such cases for a small piece of retained placenta; for a comparatively small piece may suffice to liberate fatal poisons.

I have to thank Sir Halliday Croom and Dr Haultain for their kindness in allowing me to obtain the specimens of placenta from the Maternity Hospital during their period of office there. My indebtedness to Professor Kynoch of Dundee, Professor Briggs of Liverpool, and Dr A. C. M'Master, Edinburgh, I have expressed elsewhere.

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The President said they were very much indebted to Dr Young for, he might say, his most wonderfully interesting and attractive demonstration. He had great pleasure in observing it. The first point that struck him was that Dr Young had shown that if a villus be entirely cut off from the fœtal blood supply, it could live, so long as it was in contact with healthy maternal blood. This, he thought, an interesting fact, and different from what we are generally led to believe from others.

Zweifel had long ago said that eclampsia was a disease of theories, and Dr Young had, this evening, added another to the many that already existed. It was, of course, very difficult to follow a demonstration like this for the first time. One was, of course, quite well acquainted with the infarcts that form in the ageing placenta. So far as he had followed Dr Young, he had shown that the cause of these infarcts was blood-clot,

and that they caused autolysis of the placental tissue alongside of it. He had gone a stage further and shown that the area of placenta beside the blood-clot degenerated, and toxins were formed in the diseased tissue. These passed into the circulation, and so gave rise to the eclamptic seizure. Dr Young had very materially supported his case by experiments on guinea-pigs, producing similar changes in them. Interesting though his demonstration had been, eclampsia remained a mystery yet.

With regard to accidental hæmorrhage, he (Sir Halliday Croom) was not quite so sure, but that he believed that only 50 per cent. of the accidental hæmorrhages were associated with albuminuria.

Dr Berry Hart had listened with great interest to Dr Young's demonstration, and congratulated him on the beauty of his specimens. A great many questions had been opened up—infarcts, the nature of the toxin in eclampsia, accidental hæmorrhage, and also some physiological points in regard to the villi and their attachments to the uterus.

With regard to infarcts, Dr Young held that it is a process apparently connected with the maternal tissue, and not with the villi. The other view is that it is connected with the villi, that we get an alteration in the covering of the villi which produces a clotting in the maternal circulation. The latter, the current view, was probably the more correct. To say that the villous blood-vessels are of no importance in the nourishment of the villi was probably much too strong a statement.

The specimens of infarcts were exceedingly interesting, and no doubt when one studies the infarcts, the maternal and fœtal elements are so altered that one cannot tell which is the horse and which the cart, and one might put the cart first, as Dr Young had done.

Then, as to the nature of the toxin in eclampsia, which was the title of the paper, he was specially interested in that;

he knew nothing definite as to it, and even after Dr Young's demonstration was inclined to say they were in the same position. Dr Young's theory is that one gets an infarct in the placenta, that there are toxins in this infarct, and that because this infarct is dead tissue the toxins are absorbed and eclampsia is thereby produced. But, one must ask, "Where do you get the toxins that are in the infarct?" That was the whole point. The toxins are not manufactured in the infarct; they are in the blood and tissue too, and are deposited in the clotted blood before the infarct takes place. Consequently they were no farther forward in understanding the nature of eclampsia.

The question of accidental hæmorrhage was also a complicated one. The President says you don't get accidental hæmorrhage without albuminuria. It was difficult to say what the exact position might be.

Dr Young, further, made some statements with regard to the attachment between the uterus and placenta, and it is questionable if these statements are quite correct. According to him, the placenta is very loosely attached to the uterus, resembling one glass plate laid upon another. He (Dr Berry Hart) thought it was much more strongly attached, although the spongy appearance might suggest looseness; for instance, in the first and second stages of pregnancy, although violent pains are going on, there is no detachment of the placenta.

While acknowledging the great amount of trouble Dr Young had taken, he was not quite clear as to the maternal origin of the infarct, nor did he (Dr Berry Hart) think him right in his view as to the nature of eclampsia. Dr Young clinched it by his experimental proofs. When Dr Young injected his substance, he stated he got anæmic foci in the liver. It had struck him (Dr Berry Hart) that there was a good deal of coagulation and necrosis in the liver generally, whereas in eclampsia the necrosis is in the periphery of the

lobule. This focal necrosis was also seen in hyperemesis gravidarum, although more in the centre. Wooldrige injected extracts of tissues into the circulation and got analogous results, namely, clotting in the areas whose vessels he injected, and also convulsive disturbance, if he remembered rightly.

He repeated that Dr Young's work was most excellent and he had a great deal of material to work on, but he had not surmounted the difficulty. He did not think that Dr Young could say that these infarcts were the cause of the eclampsia.

Dr Oliphant Nicholson said he had appreciated the paper very much. He thought that Dr Young had elaborated some of the suggestions that he (Dr Nicholson) had made in several papers dealing with eclampsia. The toxæmia culminating in eclampsia was due to some substance which acted on the circulation in a manner very similar to adrenalin or pituitary extract. There were many things which suggested that the placenta was concerned, directly or indirectly, with the elaboration of the toxin, and when Dixon and Taylor, in 1907, stated that the juice of the human placenta contained a principle which contracted the arteries and raised the blood-pressure, even more markedly than adrenalin, he thought it possible that these observers had really discovered the toxin of eclampsia. The anuria—the most striking clinical symptom of the disease—was undoubtedly the result of the action of some very potent vasoconstrictor which cut the arterial blood supply off the kidney, and led to intense venous congestion of the organ. The other glandular organs suffered in the same way—liver, lungs, spleen, etc.—and certainly the placenta itself would be similarly affected, and portions of its substance might well undergo necrosis. In 1909 Rosenstein wrote a paper which apparently abolished much of the interest that many people took in Dixon and Taylor's observation. Rosenstein stated that *fresh* human placenta did not contain any blood-pressure raising substance—the

Dixon-Taylor principle was, he said, elaborated in the putrid placenta only, and by the help of micro-organisms. Now he (Dr Nicholson) did not think that that assertion necessarily took away from the value of Dixon and Taylor's work, and in his remarks on the discussion of a paper read before this Society by Dr Johnstone in March 1911, he suggested that if portions of the placenta could undergo autolysis during intra-uterine life, then these powerful blood-pressure raising bodies might be developed. He thought it quite likely that the substance which Dr Young was injecting into guinea-pigs, and which caused not only convulsions, but, more important still, liver changes similar to those found in eclampsia, was formed by the autolytic enzymes of the placenta. Bits of the placenta had died, and if a dead portion, wherein these toxic bodies had been formed, happened to be in close contact with the maternal blood-stream, the circulation would readily be flooded with the poisons.

He differed from Dr Young as to the mechanism which led up to the death of portions of placenta, and thus to the manufacture of toxins in the necrosed patches. He had studied in some detail the physiological changes of early pregnancy and had no doubt whatever that some vaso-constricting substance was liberated into the blood in the early weeks of gestation, and long before any autolytic changes would be likely to occur in the placenta. Clinically the changes in the maternal heart and circulation proved conclusively that there was present in the blood some principle which caused contraction of the arterioles. The temporary grafting of the placenta—a new metabolic gland —into the maternal organism, was certain to produce a more or less profound hormonal upheaval, and that, in his opinion, was responsible for the appearance in the blood of a vaso-constricting substance just as soon as pregnancy commenced. That metabolic upset constituted the "toxæmia of pregnancy," and whether it was a trifling affair, or an extremely grave disturbance ending in eclampsia, depended, largely he believed, upon

the *personal factor* present. In certain cases vaso-constriction became so intense and widespread that portions of various glandular organs necrosed, and probably the placenta was the first organ to suffer in this way. Then the factors for eclampsia might be present. He thought Dr Young's admirable work had shed an important light on the whole subject of pregnancy toxæmias, and he wished him success in the development of it.

Dr J. W. Ballantyne said there were many points in the paper worthy of discussion, but he only wished to refer to the nutrition of the villi. Dr Young would, he supposed, admit that it was the blood passing from the villi which ultimately reached the fœtus, and must necessarily nourish the fœtus. As a matter of fact, the organs of the fœtus nearest the villi (*e.g.* the liver) were the best nourished. The blood in the fœtal villi no doubt got its supply of oxygen and other things from the maternal blood, but yet it served to nourish the villi. Dr Young said even if the fœtal blood was away it could still go on living, and had shown sections. Dr Ballantyne had no doubt that Dr Young had followed the villus in serial sections, but even then the human element of personal error could come in. In leucocythæmia the maternal blood in part of the placenta might contain less than a million red corpuscles, whilst in the fœtal villus they might find a perfect blood. In such a case the nutrition of the villus must be dependent upon the blood of the child. Again, in a case where there was general dropsy of the fœtus the maternal blood might be perfectly healthy, and yet the placenta was absolutely unnourished because the fœtal blood was defective; whereas according to Dr Young that should have been a well-nourished placenta. These seemed to him to be points on which one had to be very, very sure of the explanation before one took that view. He would like Dr Young to look well into this. He greatly admired the

piece of work as a whole thing, but he was not clear about the conclusion respecting the nutrition of the villi.

Dr W. Fordyce said one could not speak about eclampsia without bringing up a hundred points. With regard to the paper, he was very much interested in Dr Berry Hart's position. It seemed to him that Dr Young had demonstrated certain maternal changes in the placenta. In eclampsia certain changes occur in the liver and kidneys. Dr Young had brought no proof to show that the poison is derived from the placenta any more than from the liver and kidneys. He was sorry that in the experimental part he did not dwell upon this. If he took any of the injection from these thrombotic areas in the placenta, he was, of course, simply taking blood from a toxæmic woman, and one would like to know if he took blood from a healthy source and got similar results. On these points further information was required. They had not been told what substance was injected.

Dr Cowan Guthrie was very much pleased with Dr Young's remarks. As a matter of fact, in a severe wound there was practically quite sufficient poison in the destruction of the cells themselves to produce changes in the liver and kidney as well as elsewhere. If eclampsia was due in some cases to increased constriction of the maternal blood-vessels, we could see how it was that veratrum viride, which Dr Haultain had advocated, had such a specific effect in relieving this eclampsia. He quite agreed that in decomposing tissue inside the placenta one got the results produced to which Dr Young referred.

Dr R. W. Johnstone thought the paper covered such an exceedingly large area that it was difficult to discuss it. Dr Young ran counter to all the more generally accepted theories with regard to the question of infarcts. It seemed to him

(Dr Johnstone) that the intervillous space is, practically speaking, one continuous space more or less divided into areas by decidual septa. Accordingly if only one or two vessels are left patent they should be quite sufficient to supply all the villi. Another question he would ask was, how does Dr Young apply his theory to post-partum eclampsia? Twenty per cent. of eclamptic cases occur after delivery, and they have been sufficient to wreck more than one theory of eclampsia.

Dr James Young, in reply, said he was fully expecting a severe criticism, and he had not been disappointed, in fact he was rather pleased. Notwithstanding it all, he still adhered as firmly as ever to his points. In the first place, practically everything that had been said with regard to the fœtal origin of the nutrition of the villi had left out of account all the facts that bear on this question. For example, in the early ovum, where there were no fœtal vessels at all, the trophoblast was in its most active growth and was dependent entirely upon the maternal vessels. Did it live for some time on the maternal blood and then at a later date derive its nourishment from another source? If so, how did this occur? The question had just to be stated in this way for its great improbability to become evident. So, if a piece of placenta was retained in the uterus it might live for a long time.

Dr Berry Hart's remarks were interesting. He referred to the question of toxæmia and accidental hæmorrhage. The non-essential nature of the relationship between these two he (Dr Young) believed he had explained for the first time. Then as regards the way in which a dying patch of placenta could elaborate poisons, Dr Berry Hart seemed to be unaware of the fact that dangerous poisons could be elaborated by autolysing tissues. This was one of the most far-reaching questions in modern pathology. As regards the production of the liver lesions in eclampsia, surely everyone must admit

that the fact that these had been obtained for the first time experimentally, and, in addition, as the final test of a previous process of reasoning, was extremely strong confirmation, if not absolute proof, of the thesis.

II. STILL-BIRTHS' REGISTRATION.

By J. W. Ballantyne, M.D., F.R.C.P.E., Physician, Royal Maternity Hospital, Edinburgh.

The first difficulty which arises in connection with the consideration of still-births' registration is one of definition. What is registration, and what, more particularly, is meant by a still-birth? The difficulty, as will appear, is a very real and a fundamental one.

Let *registration* be taken first, for it is the more manageable of the two. Notification is not registration. Notification is giving notice or making intimation; registration implies the two acts of "declaration" and "inscription." There is registration of a death, but there is only notification of an infectious disease. In connection with births, there is in this country (Great Britain), both notification (within thirty-six hours) and registration (within forty-two days); but the former is, by the Notification of Births Act of 1907, only in force in the sanitary districts which have adopted it in the prescribed manner. In England and Wales it has been adopted in 316 out of the 1822 local government areas into which the country is divided; but these 316 areas represent nearly 18,000,000 persons out of a total population of 36,000,000. In the places which have notification as well as registration, a birth, therefore, is notified or declared twice, once within thirty-six hours of its occurrence, and again at any time within forty-two days. The persons upon whom the duty of declaring

the birth is laid differ in the two cases. In notification the task devolves upon the father of the child (whether it be a legitimate or an illegitimate one) if residing in the house at the time of the birth, and upon any person in attendance upon the mother at the time (thus bringing in the doctor and the nurse or midwife), or called in to render assistance within six hours thereafter. In registration the declaration has to be made by the father or mother if the child be legitimate, by the mother alone if illegitimate, and in their default by the occupier of the house in which to his knowledge the child was born, and upon every person present at the birth, and also upon the person having charge of this child. Whilst notification does not entail so much as registration, it is an interesting fact that in one respect (that of still-births) the Notification of Births Act of 1907 for this country goes further than the Registration of Births Act of 1874; the latter is limited in its requirements to the births of children born alive, whilst the former (in the areas, of course, in which it has been adopted) calls for declaration of birth with respect to children born alive and to those born dead after twenty-eight weeks' uterine gestation. This is a very important matter to bear in mind; it means that in the local government areas in which the Act has been adopted there is notification, although not registration, of a certain number of still-births. This brings up the definition of the word *still-birth*.

Sir James Murray's great work, *A New English Dictionary upon Historical Principles*, has unfortunately not reached the word *still-birth*, and cannot, therefore, be cited as an authority upon the definition of the term; but I notice that opposite *dead-birth* the editor has placed "still-birth" as a synonym. Dr Johnson defines *still-born* as "born lifeless, dead in the birth"; and to Webster *still-birth* is "the state of being born without life, a thing born without life." The medical lexico-

graphers of more recent times follow in the same track, the *Sydenham Society's Lexicon* saying that still-born is "born lifeless," Gould defining still-birth as the birth of a dead child, and Foster in Appleton's *Medical Dictionary* giving "dead at the time of birth" as the meaning of still-born. In Green's *Encyclopædia and Dictionary of Medicine* I did not define still-birth, but cross-referenced it to the article on the new-born infant,[1] where I wrote as follows: "Asphyxia neonatorum, apnœa neonatorum, still-birth, or apparent death of the new-born, is a condition of the infant at birth in which there is a stoppage of the functions of animal life, with absence of respiration, and absence, or rather weakness, of cardiac action; a still-born child is not necessarily a dead child, but the condition may pass into death." My reason for expressing the condition of still-birth in the above phrases is easily stated; every obstetrician meets with cases in which, although the infant is born apparently lifeless, in which it neither breathes nor moves, he is nevertheless able by appropriate means to resuscitate it (I use the word "resuscitate" here, but must criticise its employment in this connection later); it was still-born, and yet it now lives; consequently I do not regard "born dead" as a correct definition of still-born. The whole difficulty arises on account of the lack of a proper realisation of the true nature of ante-natal as compared with post-natal life, and to this matter I shall return after the various legal and administrative statements of the meaning of still-birth have been taken notice of.

By far the most valuable contribution which has been made to the proper understanding of still-births and their registration of recent years is that contained in the Report of the Special Committee of the Royal Statistical Society on Infantile Mortality, published in December 1912.[2] Dr

[1] Green's *Encyclopaedia of Medicine and Surgery*, vii., p. 57.

[2] *Journal of the Royal Statistical Society*, lxxvi., pp. 27-87, 1913.

Reginald Dudfield's paper on the Report,[1] and the discussion which followed it also contain much that is of interest for the elucidation of the subject. One feels that a great advance has been rendered possible in regard to legislation upon the subject of still-births, their registration, their investigation, their causation, and ultimately their prevention. The Committee addressed inquiries to 136 registration officers all over the world, and received replies from 103. The countries, colonies, and states which did not reply were Greece, Portugal, Russia, Servia, China, Brazil, Chile, Guatemala, Honduras, Mexico, certain British possessions (including British Columbia, British Honduras, Cyprus, Falkland Islands, Fiji, and Quebec), some German states (viz., Baden, Brunswick, Mecklenburg-Schwerin, Oldenburg), and twelve of the states of the United States (including Alabama, Arkansas, Delaware, Florida, Kentucky, Louisiana, South Carolina, etc.). The questions asked had to do with birth-registration, death-registration, and the registration of still-births, so that on these matters it may be taken for granted that the Committee's report puts us in full possession of all the facts for the whole civilised world with the exception of the places above named.

I cannot spare space to discuss the information gained regarding the registration of births, although it may be noted, in passing, as a somewhat surprising fact that this practice is not compulsory in Finland, Iowa, Mississippi, and North Carolina and Virginia (outside town limits); but I shall pass at once to the replies regarding still-births and their registration.

The registration of still-births is not compulsory in the following countries and colonies:—England and Wales, Scotland, Ireland, Gibraltar, Cyprus, Hong-Kong, Gambia, Orange Free State, Sierra Leone, Jamaica, New Brunswick, New South Wales, New Zealand, Queensland, South Australia, Tasmania, Victoria, Denmark, Mississippi, New Mexico, North Carolina,

[1] *Journal of the Royal Statistical Society*, lxxvi., pp. 1-26.

and Virginia. In other words, it is not compulsory in Great Britain and in certain of her dependencies whose laws have been founded on her model, in Denmark, and in a few of the individual states of the United States of America. But with regard to Denmark, it is noted that "although not prescribed by law, the registration of still-births is generally regarded as compulsory." In some of the states of the United States, too, although registration is not compulsory, there are circumstances which modify the effect of the omission. Thus, in New Mexico the absence of registration seems to be due to bad wording of the law, and the burial of still-born children is not possible in "legal cemeteries" without death certificate. In North Carolina, also, still-births are entered on death registers, for burial permits are necessary. These additional facts serve to throw into clearer light the outstanding result of this part of the inquiry, viz., that it is in Great Britain and the countries whose laws have been framed on hers that still-births are not compulsorily registered. It has also to be noted that in Malta, Ceylon (in part), India, Strait Settlements, Cape of Good Hope (in a degree), Mauritius, Natal, Transvaal, Alberta, Bermuda, British Guiana, Manitoba, Newfoundland, Nova Scotia, Ontario, and Western Australia, there is either registration of still-births or else an equivalent, *e.g.*, they are regarded as "births." It is fair, therefore, to say, and to say with emphasis, that, granting the usefulness of still-births' registration, Great Britain and Ireland have been singularly dilatory in taking legislative steps to acquire powers to discover the still-birth rate in the population living in them.

One turns with hopefulness to the numerous countries which have compulsory registration of still-births in order to obtain from one or other of them a satisfactory definition of the word still-birth; but one is doomed to disappointment, for the Report of the Special Committee of the Royal Statistical Society contains the following surprising statement: "So far

as can be ascertained no legislature has, up to the present, enacted any definition of still-birth, and the only guidance afforded to those required to declare and inscribe such births is that afforded by regulations made by the various statistical offices." Even the interpretations adopted for registration purposes differ so widely among themselves as to introduce great confusion into the subject and to make comparative statistics impossible of obtainment, or at least useless for practical application. Here, then, is a very strange state of affairs; most of the civilised countries of the world make the registration of still-births compulsory, yet none of them has enacted any definition of still-birth, and there is great diversity of usage amongst them as to the meaning of the terms as officially applied in the working of registration; nevertheless the practice of registration is going on in these countries and has been going on in some of them for a long time. It is no wonder that the first conclusion drawn by the Special Committee of the Royal Statistical Society is that "it is desirable that an international definition of 'still-birth' should be adopted."

Some of the methods in which the difficulty of absence of precise and legal definition is got over may be referred to. In the first place, many countries simply require all *births* to be registered, and so of course still-births are, so to say, caught in the meshes of the registrational net; but there is reason to believe that all are not so caught. This is the practice, for instance, in Newfoundland, Nova Scotia, Belgium, and California; but obviously this plan of massing together the births of living and dead infants cannot be looked upon as ideal. In the second place, other countries supply their statistical or registration officers with regulations regarding still-births; but it must at once be admitted that these differ so widely both as regards the state of the infant which is to be called still-born and the age of ante-natal life at which the fœtus shall have arrived in order to come within the scope of registration that confusion

rather than perspicuity has been their effect. One cannot help pitying the officials in their task, and deploring the necessarily unsatisfactory nature of the results of their labours.

Some examples will serve to illustrate what has been stated above. In Denmark, "every fœtus coming into the world without any sign of life during or after the 28th week of pregnancy is counted as a still-birth; an embryo expelled (*dead*) before the 28th week is reckoned as an abortion." In Hungary, "children born after seven months' gestation without signs of life are registered as still-born; those born earlier are ignored." In Japan, a very early date is fixed, and "registration of a fœtus born after four months' gestation is required." A still-born child in Switzerland is one "dead before birth or dying during birth." In Ceylon, "the term still-birth means a child born after the 28th week of gestation as dead, or apparently dead and not called back to life"; and one cannot but feel that in this attempted definition there are signs of light. In Nova Scotia, "a 'still-birth' is a child born dead." In Western Australia, "a still-born child means any child of seven months' gestation or over not born alive"; but for the purposes of the Registration Act (1894) a still-born child is "deemed to have been born alive and to have subsequently died." In Missouri, children "dead at birth" are reckoned as still-born; so also in Montana. In Massachusetts, a still-birth is a child "born dead"; and so also in Minnesota. In Nebraska, it is "a child who has not breathed"; and in Iowa nearly the same words are used; whilst in New Hampshire, it is "a viable child not born alive." In Norway, the interpretation adopted for registration purposes is that "embryos born without signs of life after the commencement of the eighth month" are counted as still-births; whilst in Sweden, "a child is described as living-born if it breathes after birth, as still-born (dödfödd) if it manifests no signs of life after birth." In Egypt, a still-born child is "a child not breathing at the time of birth"; in Gibraltar, "a

child who was not born alive"; in India, "a viable child reported to have been born dead"; in Gambia, "one who has died before delivery, *i.e.* in its mother's womb"; in the Mauritius, "a child of seven months' gestation (or more) which died before delivery"; in Jamaica, "a child who has had no separate life from that of its mother." The interpretations in the German states also differ. In Anhalt, a still-born child is "one showing no sign of life prior to complete separation from the mother"; in Lubeck, it is "a child of six or more months' gestation, and 32 cm. length, born dead"; in Hesse, it is "one born dead or dying during birth after six months' gestation (or longer), the fœtus being not less than 30 cm. long"; in Saxony, it is "an embryo born dead after a gestation of six months or longer"; and in Saxe-Coburg Gotha, the law distinguishes between a child born dead and one dying in birth.

So far the interpretations have rung the changes on "dead-born," "dying in birth," and "without signs of life," whilst some have introduced ante-natal age limits (4 months, 6 months, 7 months, 28 weeks, viability), and others have even mentioned the length of the fœtus necessary for registrational purposes. All these regulations are confusing and complicated enough; but the full limit of perplexity and obscuration, even to stupefaction, is reached only with the regulations in New York, France, Belgium, Holland, Spain, Prussia, and our own country. The New York interpretation is comparatively harmless, although it introduces a new characteristic into the matter, viz., that of sex: it is, "a child dead after having never breathed and having attained sufficient development to determine sex." Obviously if this interpretation, which is the one "prescribed by the State Department of Health," be strictly acted upon it will carry the earliest age for registration pretty far back in ante-natal life. The same element of the recognition of sex crops up in a decision of the Court of Besançon, in France, in 1844, from which it appears that "the registration of

a still-birth is held by French jurisprudence to include, not only children who have been the full term in the womb, but all such as are born sufficiently formed for it to be possible to declare the sex." In France at the present day the interpretation of still-born child for registration purposes is "a child born dead or dying before the declaration of birth." Now the last phrase of the foregoing interpretation introduces new difficulties and a certain degree of inaccuracy into statistics; and this applies to Belgium, Holland, and Spain, as well as to France, for in all these countries the lists of the still-births must necessarily include some children dying between birth and declaration or registration. Further, the regulation has necessitated the coining of a new term for the children born alive but dying before declaration, and Bertillon has called them "dits mort-nés," the others being simply "mort-nés." Since the time allowed for registration in these countries is three days, and since a good many babies die in the first hours of life, it is probable that a considerable proportion of the babies registered as still-births were really not so in the sense of the word as used in other parts of the world. In Belgium, the confusion has been in some measure cleared up by calling the one class "les enfants présentés sans vie," and the other the "mort-nés" proper. In Italy also, and in Roumania, there is no distinction for registration purposes between a still-born child and one born alive and dying before registration of birth. In Prussia, there are three classes of births called still-births: there are full-term children born dead, there are embryos born dead after six months' (180 days') gestation, which are more than 32 cm. in length, and there are embryos closely approximating to the conditions just named.

In the British Isles, although the registration of still-births is not compulsory, there is nevertheless an official interpretation of still-birth. In England, "a live-born child for registration purposes is defined as one that lived (for however short a time)

after complete expulsion from the body of its mother; all others are to be regarded as still-born." Or, putting the definition affirmatively, "a child is deemed to be still-born when after being completely born it has not breathed or shown any sign of life." In Scotland, "if the child has lived, *i.e.*, had separate existence from its mother, and has breathed, both birth and death are recorded; otherwise no entry falls to be made in the registers." In both countries there is the Notification of Births Act, which local authorities may, with the consent of the Local Government Board, adopt, and by which when so adopted every birth, even although it be a still-birth, must be notified within thirty-six hours. The interpretation of still-birth in connection with this Act is the birth of a dead child which takes place at or later than the 28th week of pregnancy. Reference may here be made to two definitions of still-birth, which one may term private ones. Dr Reginald Dudfield's is: "a 'still-born child' means a child born after a period of gestation of not less than seven lunar months (twenty-eight weeks) whose heart has ceased to function before the whole of the body (including the head and limbs) of such child has been completely extruded from the body of the mother; and still-birth means the birth of a still-born child." The other definition is that of the Obstetrical Section of the Royal Society of Medicine: "a still-born child means a child which measures more than thirteen (13) inches in length from the top of the head to the heel, and which, when completely extruded from the body of the mother (head, body, and limbs, but not necessarily the after-birth), exhibits no sign of life by crying, or breathing, or by pulsation in the cord at its attachment to the body of the child, or by beating of the heart."

What has been stated in the above paragraphs is an index of the amazing confusion into which the whole subject of still-births and their registration or notification has passed, Indeed it is not a full revelation of the whole entanglement.

for I have not touched upon the diverse procedures which have been adopted in different countries as to the statistical place accorded to still-births; as a matter of fact, they are entered in some lands as births, in others as deaths, in others as both births and deaths, and in yet others separately from births and deaths. In the great majority of the countries the last-named place is that given to them. But enough of the demonstration of the registrational and definitional labyrinth into which the subject of still-births has got. Is there any Ariadne thread which can be used to get the investigator out, or must he cut his way through this legislative and lexicographic jungle? I venture to think that by the way in which the confusion was introduced the path of escape may be found. Let me explain.

The greater part of the confusion in the matter of still-births is due to the lack of correct views regarding the nature of ante-natal life and the peculiarities of the physiology of the fœtus, and much of the perplexity in which one finds oneself in trying to elucidate the subject may be got rid of by gaining right notions respecting the life before birth, and by employing them in the revision of the terminology of still-births, live-births, dead-births, and the like.

To begin at the beginning, life does not commence at birth but before it. A special kind of life begins then, which may, for the sake of distinction, be called post-natal; but the fœtus has already been alive biologically, if not legally, for several months, and the embryo before it had a vitality so extraordinary as to enable all the complexities of organo-genesis to be exhibited in a marvellously short number of weeks. This life of the fœtus and embryo I have called ante-natal, and have claimed for it a reality and an importance which have not always been accorded to it by others. I began using the word in association with pathology as a convenient one to designate the many morbid states which had their origin

before birth. As far back as 1892 I published the first volume of a book entitled *Diseases and Deformities of the Fœtus*, and gave to it the sub-title of *An Attempt towards a System of Ante-Natal Pathology*. I might, of course, have called my subject pre-natal pathology or medicine, but for some passing or accidental reason which I have now forgotten I chose the word ante-natal. It had the high sanction of Sir James Murray's *New English Dictionary*. It had been used in 1817 by Shelley, who spoke of "memories of an ante-natal life"; by Kingsley in 1848, when he referred to "heaven my spirit's ante-natal home"; and by Farrar in 1879, when he described the "ante-natal predilection for Israel and detestation of Esau"; it could not, therefore, be wrong for the physician to employ the term and follow the glossarial example of the poet, the novelist, and the clergyman.

Ante-natal life has, in comparison with and in contrast to post-natal life, this one great differentiating character, its respiration is a tissue one, made possible by the placenta from which oxygen is drawn over from the maternal blood and through which carbonic acid and other things are sent over into the mother from her fœtus; the respiration of post-natal life is a pulmonary one and an atmospheric one. There are other differences between the two modes or forms of life which will occur to anyone who thinks for a moment, but they are far less striking than the resemblances, for in both ante-natal and post-natal life there is cardiac action and circulation, in both there are free muscular movements, in both there is activity of the nervous system, in both there is blood formation; and in both, the liver, kidneys, spleen, intestine, ductless glands, and the like are physiologically active, although of course the particular circumstances of ante-natal life call for a far smaller exhibition of functioning than do those of post-natal existence. Save in the matter of respiration, the differences in the two lives are those of degree rather than

of kind. Of course there is a difference too between the ante-natal life of the early months of intra-uterine existence and that of the later months, but this difference also is more one of degree than of quality. I am aware that such things as viability and its date are spoken of; but I lay no great stress upon the former from the standpoint of still-births, and I affirm that the latter cannot be fixed because every invention for the keeping alive of prematurely born infants and every improvement in infantile hygiene must push back the date of viability nearer to conception.

There is, however, a very great difference between the life of the first weeks after impregnation and that of the months which follow it, for in the former it is embryonic, formative, or developmental activity which is strongly in evidence, whilst in the latter it is life and growth very much the same (except, of course, for the pulmonary respiration) as they are found post-natal. Further, the embryo in its decidual membranes is quite easily distinguished from the fœtus with its placenta, and if it be desired to introduce a classification at all into ante-natal life, it will be better to divide it into embryonic and fœtal rather than into viable and non-viable.

But it may be asked, when does ante-natal life really begin, what is its actual *terminus a quo?* I regard it as commencing when the spermatozoon effectively penetrates the ovum and sets in action the long series of events which end at birth. If a later date is attempted all the ambiguity regarding quickening, stirrage, feeling life, and the rest is around us, and difficulty is everywhere. To quote from a recent article by A. E. Crawley[1] on fœticide, "modern biology holds that the zygote is a new individual from the moment, not of 'conception' in the vague and popular sense, but of penetration of the ovum by the spermatozoon; and modern legislation holds the same view, but is less severe upon fœticide than upon infanticide." Tertullian,

[1] Crawley, A. E., *Encyclopædia of Religion and Ethics*, vi., 56, 1913.

many hundreds of years ago, went even further, when he said, "He also is a man who is about to be one; even every fruit already exists in its seed." Individual ante-natal life begins at impregnation. There is a sort of vitality before that, the life of the sperm cell and the germ cell among the other cells of the body; but it lacks the individuality of the life of the zygote, and need not be dealt with now.

For nine months, therefore, before birth there has been individual life. Further, death may occur during this life as well as during post-natal existence, and the great sign of this death is cessation of cardiac action; there are other signs of course, even rigor mortis may occur, but stoppage of the heart is the decisive indication of ante-natal death. There can be no help got from cessation of pulmonary respiration, for the unborn infant does not respire through the lungs. The outstanding distinction, therefore, between ante-natal death and post-natal death is that in the former there is no cessation of pulmonary respiration, whereas in the latter there must be. Ante-natal life lacks something which post-natal life has, viz., pulmonary respiration; consequently ante-natal death likewise must lack a characteristic feature. Ante-natal life has placental circulation as the analogue of post-natal pulmonary respiration, and ante-natal death has the stoppage of placental circulation as its characteristic sign. The other marks of difference are, so to say, accidental; they are due to the ante-natal environment, which does not suffer the presence of putrefactive microbes, and so, whilst it permits maceration, does not allow putrefaction (unless indeed by man's interference). These macerative changes are important because they are easily recognisable; and when recognised they enable one to say that ante-natal death has taken place some days previous, it may be weeks or even months, but it is certainly days. It ought perhaps to be added that now and again cases of the birth of so-called macerated living fœtuses have been recorded; in all these instances the maceration, I

believe, consisted simply in desquamation of the cuticle from some morbid state of fœtal life.

With these facts in full view, it is permissible to attempt the construction of some definitions; and, first of all, how is live-birth to be interpreted? The present definition or interpretation for registrational purposes is that a "live-born child is one that lived (for however short a time) after complete expulsion from the body of its mother"; and live-birth is "the manifestation of some certain sign or signs of life by the child after it is completely born." So far so good; but there are three expressions involved in these statements which are themselves in need of definition, and of these three "child" is perhaps the least definite. There are two extremes; any product of pregnancy without reference to its ante-natal age may be called a "child," or the term may be restricted to fœtuses born not earlier than the 28th week of pregnancy. If one adopt the former, one is face to face with the terminological absurdity of calling an embryo of 1 cm. long in the decidual membranes a "child," whilst if one takes the 28th week as the earliest age one is excluding fœtuses which may quite well on occasion live and grow into adult men and women. If, again, viability be chosen as the delimiting age, one is at once faced with the difficulty (to which reference has already been made) that every improvement in the construction of the couveuse and every advance in the feeding of the premature infant may alter the age of viability and make it earlier. It is necessary, however, to fix upon a date for registrational and other forensic purposes, and personally I should prefer to say twenty-four instead of twenty-eight weeks as the earliest age to which the name child is applicable, but I am ready to accept twenty-eight weeks for the sake of uniformity. In the next place, "completely born" requires definition. It is usually taken to mean complete expulsion of the child from the body of the mother, but not entire separation; in other words, it is

not necessary that either the umbilical cord should be divided or the placenta expelled. A child, therefore, is completely born when its head, trunk, and limbs are all outside the vulva; and to this meaning I do not think exception can be taken, although it may and does introduce ambiguity and indefiniteness into the medical evidence in infanticide trials, and possibly stands in the way of conviction in a few undoubtedly criminal cases. With regard to the significance of "certain sign or signs of life," there is again some confusion due to a want of appreciation of the characters of ante-natal life. The signs of life usually considered are four in number: breathing, crying, movements, and heart-beat or its resulting pulsation (in the cord, at the wrist, at the ankle, and elsewhere). Obviously the first two are purely post-natal in their time of occurrence, neglecting the few instances in which a child has cried *in utero*, or tried with disastrous effects to itself to breathe therein; the other two (movements and heart-beat or vascular pulsation) are ante-natal. Breathing, therefore, is, speaking generally, the best sign of life to use as a test of live-birth, for it is the characteristic indication of the commencement of post-natal life; crying is not so good, for the child may lie breathing for some time before it cries, whereas it can hardly cry without breathing; the other two (movements of the limbs, face, etc., and heart-beat and resulting vascular pulsation) are of less use for the present purpose, because they were already in existence in ante-natal life and could be detected then. As a matter of fact, there is a curious paradox here; for if the pulse in the umbilical cord be regarded as the sign of live-birth (as the Obstetrical Section of the Royal Society of Medicine seems to suggest), then there is the disturbing fact that as soon as pulmonary respiration is fairly established the pulsations in the cord lessen in strength and diminish in number until they stop altogether; so that the commencement of one sign of live-birth is accompanied by the extinction of another.

Applying, now, these considerations to the defining of the term live-birth, it seems to be that there are really two varieties of live-born children—those which after complete expulsion from the body of the mother show the signs of ante-natal life alone (and consequently soon perish, these vital processes by themselves being insufficient to maintain life in the absence of the placental circulation), and those other which exhibit the signs of ante-natal life and assume in addition the evidences of post-natal life (breathing or crying, the indications of pulmonary respiration). I am interested to find that in Queensland the definition (for registration purposes) of "living person" is as follows: "A child becomes a person capable of being killed when it has completely proceeded in a living state from the body of its mother, whether it has breathed or not, and whether it has an independent circulation or not, and whether the navel-string is severed or not." In this somewhat diffuse, periphrastic, and circumlocutory definition the Queensland officials show a certain acquaintance with the facts of ante-natal life which is both unexpected and highly encouraging.

What, next, can be said about such a term as dead-birth? I am personally of opinion that the words dead-birth and dead-born should be strictly reserved for the cases in which the child has died in ante-natal life, that is to say, anterior to the commencement of labour. The "dead-born child" I should regard as a child which after complete expulsion from the body of its mother shows that it had ceased to exhibit the signs of ante-natal life at some period prior to the commencement of labour; and, if thought necessary, I should be prepared to add, "but posterior to the 27th week of ante-natal existence." Of course, the great sign of ante-natal death is to be looked upon as maceration. I do not stay here to condemn the most misleading term "putrid" as given to a dead fœtus which is macerated but in no degree putrefactive; let it be given, if thought desirable, to the dead fœtuses to which the germs of putrefaction

have gained access in the uterus, and in which intra-uterine putrefaction in all its loathsomeness and offensive effluvium is so nauseatingly evident. It is a rare occurrence nowadays, but it could for registrational purposes be introduced after the term macerated, thus, "macerated (and putrid)."

I feel disposed to place by themselves the children which enjoy ante-natal life up to the time of birth, but which lose their ante-natal vitality in the process of birth before they have had the chance of adding to its signs those of post-natal life. These are the children brought alive to the birth, but not born alive. I refer to the craniotomised child, soon to become, I trust, as unknown as the whalebone fillet in operative obstetrics; to the child dying by reason of pressure on the umbilical cord; to the fœtal victim of placenta prævia or accidental hæmorrhage; and, in a word, to all children whose hearts could be heard at the beginning of labour, but which show no signs of ante-natal life at the moment of birth. If necessary I should call them dead-in-birth children, and the condition intra-natal death. I am willing, however, for the sake of simplicity in a subject overburdened already with complexity, to group them with the dead-births, as above described, adding to the last clause of the definition the words "during or," when the clause would read "during or at some period prior to the commencement of labour." At the same time, there are good reasons for trying to make the distinction between ante-natal and intra-natal deaths, and the absence of macerative changes in the latter makes the separation possible.

Next there are the still-births as I think they should be defined. I am well aware that what I am now about to propose is revolutionary, but I am convinced that the proposition should be made before the drafting of a still-births' registration Bill makes it, if not for ever, at anyrate for a very long time, impossible to re-open the subject. Why, for instance, should we not define the still-born child as one expelled completely

from the body of its mother, exhibiting one or all the characteristic signs of ante-natal life (movements, heart-beat, pulsations, etc.), but failing to assume one or all those of post-natal existence (pulmonary respiration, crying, etc.). Of course, if by medical means or other plans of resuscitation it did assume the signs of post-natal life, it would pass into the category of the live-births. It may be suggested that two groups would be needed, one containing the still-born children happily resuscitated, and the other those in which the still-birth passed into post-natal death; but this would necessitate a time limit beyond which it would be regarded as hopeless to continue resuscitative measures, and so confusion would be introduced. The difficulty perhaps is more theoretical than real, for none of us, I suppose, continues the methods of resuscitation after the child's heart has ceased to beat.

I have used the terms resuscitate and resuscitation above, but it must be borne in mind that they are not quite correctly so used in this relationship. "Resuscitate" means to restore to life, to revive, or to renew; but in these infants, born apparently lifeless, it is not so much a restoration to life as a substitution of one form of vitality for another, a successful transformation or transference of ante-natal life into post-natal existence. Nature usually carries the one form of life over into the other with no jar or hiatus; before ante-natal life with its placental circulation has well ended, post-natal life with its pulmonary respiration has begun. But this is not constant, and the obstetrician has to step in and try to do what nature has failed to accomplish, *i.e.*, to replace ante-natal by post-natal life. For the same reason "re-animate" is hardly the right word, and although I propose "trans-animate," it is not quite satisfactory.

The suggestion which I have made above is simply the outcome of a more careful estimate of the real nature of birth. Birth is the transition from a state of ante-natal life with its

characteristic phenomena into one of post-natal life with its additional phenomena. In ordinary circumstances the transition is accomplished with wonderful smoothness considering the greatness of the change involved, and to the eyes of the obstetric watcher there is little to be observed save the commencement of rhythmical movements of the chest-wall, whilst upon his ears falls the soft sighing of the respiration or the more easily audible cry of the new-born. Yet in these few minutes changes of a very intimate and fundamental kind are going on inside the infant's body. The characteristic signs of ante-natal life, it is true, continue, and the infant outside of the womb moves its body and limbs as he did inside, while his heart goes on beating and drives blood through the body; but the blood-stream is changing its distribution, and instead of having a main current passing to the placenta it has now a strong flow going to the lungs. This change necessitates rearrangements, and in time is accompanied by structural alterations, closure of umbilical and hypogastric vessels and of the ductus venosus and ductus arteriosus and foramen ovale, and dilatation of the pulmonary vessels and tissues. It is also accompanied by a novel phenomenon, pulmonary respiration, and the child adds to its ante-natal life this peculiar manifestation of post-natal existence. Perhaps it is less novel than one is inclined to think, for there is some evidence that even *in utero* in the last weeks of that particular life there are slight rhythmical movements of the chest walls suggestive of and preparatory to the regular rise and fall of the thoracic framework which distinguish post-natal life. The unborn child, it is just possible, is exercising himself with a sort of preliminary course of gymnastics for his great post-natal respiratory feat. Of course, other changes or modifications of function follow: stomach digestion begins and intestinal processes become more active, although there is evidence that these parts are not quite inactive in fœtal life; the kidneys, too, either increase their

typical function or make a beginning; a more steady and complete circulation of well-aerated blood flows through all the outlying parts of the body, whilst the liver receives a less generous share; and there are other modifications or expansions of function in connection with the sense organs, the skin, and the brain which a closer scrutiny reveals. In a phrase, the living fœtus becomes a living infant; the ante-natal life has put on the post-natal.

Thus, there is birth, the transition from one form of life to another, from ante-natal existence, with its less intense but sufficient placental economy, to post-natal existence, with its abundant pulmonary opportunity; from pre-natal dependence upon the maternal tissues to post-natal partial or complete independence of them (according as the mother suckles or does not suckle her child); there is the death also which is ante-natal in time and macerative in result, and the death which is post-natal in time and is accompanied by all the ordinary post-mortem signs of putrefaction, and between the two there is that state of hesitancy in the assumption of the characteristic phenomena of post-natal life to which I desire that the term still-birth should be restricted. In this state of still-birth as thus defined the child is not dead ante-natally, for it retains one at least of the signs of ante-natal life (the fœtal heart-beat and pulsation), although it may have lost the other (fœtal movements), and so lies "still"; neither is it alive post-natally, for it has as yet failed to assume any of the characteristic signs of the fuller life which is after birth, and it does not breathe or cry; surely it is wise not to call it a dead-birth so long as there is a legitimate hope that appropriate treatment may fan the small spark of ante-natal life into the strong flame of post-natal existence; surely it is preferable to call it what it precisely and exactly is, temporary stillness at birth, not the definitive stillness of death. The intra-natal deaths, the cases in which the fœtal heart is beating at the beginning of labour

but has ceased before complete expulsion of the child has occurred, may, for simplicity, be grouped with the ante-natal deaths, for in the strict sense of the term they are ante-natal. There are, therefore, three classes of occurrences: the ante-natal deaths, in which the signs of ante-natal vitality have been extinguished before the complete expulsion of the child from the mother's body; second, the post-natal or infantile deaths, which take place after the child has been born alive, after it has successfully taken on the peculiar and characteristic signs of post-natal as distinguished from ante-natal vitality; third, the cases in which the ante-natal type of life is maintained for a short time after complete expulsion from the mother's body, in which the assumption of the characters of post-natal life may not take place, and in which, therefore, ante-natal life may give place to post-natal death without the interim establishment of post-natal life. The first class should be called ante-natal deaths or dead-births; the second class may be grouped with deaths in general or with infantile deaths in particular, although from the present standpoint they are post-natal deaths; and to the third class the name of still-births can appropriately and correctly be given, if for no other purpose than to indicate the presence of the hope of establishment of post-natal life which is inherent in them. Of course, when the still-births are happily carried across from ante-natal into post-natal life after the temporary check in the transition which they have suffered, they will be called still-births which have become living infants.

Little space is left for the discussion of the necessity for the compulsory registration of these ante-natal and intra-natal fatalities, but fortunately little space is needed for this purpose. Great Britain and Ireland, with a few of the British dominions across the seas, stand at present almost alone among the civilised nations of the world in making no registrational attempt to discover how many lives in their populations are

lost before and at birth. What the majority of the nations of the world have, what India, the Cape of Good Hope, and most parts of Canada already possess, surely ought seriously to be regarded as desirable for the rest of the British Empire, including the Mother Country and the great Continent of Australia. This is one of the many strange effects which the falling birth-rate has had; so long as there was what Tennyson called a "torrent of babies" rushing in upon the land, there was less need perhaps to give thought to the thousands of individual drops that were lost as spray, but now there is an inclination to conserve the whole of the supply and to bring every life to the birth. Obviously the first step must be to discover what the loss is. Other nations have some information on this point to guide them. At least they know something of the number of lives lost between the sixth or seventh month of pregnancy and the full term. They have, however, with one or two possible exceptions, absolutely no registrational knowledge whatever of the numbers of the ante-natal lives which are lost before the sixth month of pregnancy; abortions are taken no legislative notice of. With the exception of the information made available by the Notification of Births Act, there is in Great Britain no means of finding out the ante-natal death-rate between the 28th week of pregnancy and the full term, and, with the exception of some returns coming in from certified midwives there is no means of gauging the number of abortions, although every doctor knows how prevalent they are.

One very small advantage may accrue to this country from its backwardness in the compulsory registration of ante-natal deaths, viz., the opportunity of improving upon existing methods and of benefiting from the mistakes other nations may have made. For instance, let there be no registrational loop-hole left whereby the infants who die between birth and the hour or day of declaration and inscription shall be liable to

appear amongst the dead-borns; there must be no confusion here similar to that between the "mort-nés" and the "dits mort-nés" of France and Belgium.

Let us also get rid of the practice of regarding still-birth as synonymous with dead-birth; it is neither etymologically correct nor registrationally desirable. Let us call the child whose ante-natal life ceases at any time between (let us say) the 24th week of pregnancy and its complete birth from the mother's body a dead-born child, and let the birth of such an one be termed a dead-birth. Let all these be compulsorily registered. Let the interruptions of pregnancy prior to the sixth lunar month (24th week) be called abortions or miscarriages, and let notification of such be made optional for the various sanitary districts of the country. Let the still-births, as I have defined them, be entered twice, first as still-births, and second as either infantile deaths or as ordinary births according as the attempt to animate them has failed or succeeded. Let us at least recognise the existence and reality of ante-natal life to the extent of inscribing it. In this way it will become possible to know at least the proportion which the lives which are lost before and at birth bears to those which are lost immediately or soon after birth, as well as to have some idea of the chances of survival of still-born babies (as I have defined them).

I hardly think it would be wise at first to demand in the registration form the cause of death, far less than of the miscarriage; but it would be of the greatest benefit to have the sentence "cause of ante-natal or intra-natal death, if known," printed there. At first few medical men would be able, even if willing, to give this information; but some would give it to the best of their ability; all would become accustomed to the routine even if unproductive interrogation regarding the causes of such fatalities; and in time, with an extension of our available knowledge of ante-natal pathology, and an increase in our facilities for making post-mortem

examinations upon the products of pregnancy, it would be possible to make the clause obligatory. So, too, with the miscarriages; a phrase such as "probable cause of the abortion" could be inserted into the notification forms. Let our maternity hospitals with their greater resources in diagnosis and greater knowledge of the morbid anatomy of ante-natal life lead the way in all these matters.

But, in addition to yielding some indication of the amount of life lost before and during birth such a Registration Act would be of immense service in two other directions. During the past ten or fifteen years a great reduction has been made in the infantile death-rate for the first year of life, and it is generally recognised that the improvement in the chances of survival in these early months is largely due to the removal of causes of disease (*e.g.* diarrhœa) which come into operation after birth by bad hygiene and faulty feeding. It is also thought that the possible diminution in the infantile death-rate by the removal of these preventable causes has for some places nearly reached its limit. If, therefore, there is to be a further reduction it must be by attacking the causes of post-natal death which are in action before birth or are brought into play at birth, viz., the ante-natal or intra-natal causes of infantile mortality. At present, as Dr Dudfield admits, very little is known of these ante-natal morbid causes which throw their malign influence forward into post-natal life; but from observations which have been made in New York[1] it has been calculated that no less than 35 per cent. of the total mortality under one year of life is due to congenital diseases, and these usually prove fatal in the first month. If this be so, or if it be even an approximation to the truth, it is easy to see that anything which increases our knowledge of these ante-natal causes will be of immediate service in helping us to overcome the congenital causes of infantile mortality. Then, again, by

[1] *New York Medical Journal*, xcviii., p. 383, 1913.

such a Registration Act we shall learn more of the pathology of ante-natal life itself, and so a brighter light may be thrown into the dimly obscure region known as ante-natal hygiene; thus in time will come the power of dealing with the life which is before birth in such a way as not only to prevent ante-natal deaths and miscarriages (which, of course, are ante-natal deaths, family deaths, we may say, without following funerals), but also to encourage the coming to the birth of strong and healthy infants. For all these reasons a Still-births Registration Act is needed. It will perhaps throw a fresh burden upon the medical practitioner, but legislation is not without means of ameliorating the oppressiveness of such a weight, and, further, our continental confrères have been bearing this additional professional obligation for many years.

Finally, I may say that what is really needed is not so much a Still-births Registration Act (as I have defined still-births), but an Ante-natal Deaths Registration Act. I venture to think that the latter term more correctly indicates what is lacking in our vital statistics and what is the real wish of our social reformers and ante-natal hygienists.

My leading conclusions, therefore, are as follow:—

1. That, in view of the existing confusion in the terminology of still-births and allied states, an attempt to reach uniformity of definition should be made.

2. That a Still-births or, better, an Ante-natal Deaths Registration Act is greatly needed in this country.

3. That the definitions of dead-birth, live-birth, still-birth, and abortion should be so constructed as to be in accordance with the physiology of the time of life dealt with.

(*a*) For *dead-birth* I suggest the complete expulsion from the maternal birth-canals of a child which during or before birth has lost the characters of ante-natal life, especially heart-beat, arterial pulsation, and movement.

(*b*) For *live-birth*, the complete expulsion from the maternal birth-canals of a child which, whilst it loses pulsation in the cord, adds on to the other characters of ante-natal life the signs of post-natal vitality, viz., pulmonary respiration and crying.

(*c*) For *still-birth*, the complete expulsion from the maternal birth-canals of a child which, whilst continuing to exhibit one or more of the signs of ante-natal life (heart-beat, arterial pulsation, movement), fails for a time to assume those of post-natal life (pulmonary respiration, crying, etc.), and then either loses even the characters of ante-natal life or is successfully resuscitated (transanimated), and assumes those of post-natal existence.

(*d*) For *abortion* or miscarriage I suggest the termination of ante-natal life before the end of the sixth lunar month by the expulsion of the uterine contents. A *premature birth* is expulsion of the uterine contents after the sixth lunar month, but before the full term; and it may be a dead-birth, a live-birth, or a still-birth.

The President said they were under a great obligation to Dr Ballantyne for ventilating this subject.

(It was agreed to hold the discussion over till the next Meeting.)

Meeting VI.—May 13, 1914.

Sir Halliday Croom, *President, in the Chair.*

I. At the request of the *President*, the *Secretary* read a letter from Dr H. M. Church, presenting a Presidential chair to the Society.

The chair is decorated with the crest and motto of the Society—viz., the beautiful head of Lucina (or Juno), the goddess who presided over marriage and childbirth, with an olive branch and young olives on either side, intertwined with the motto, "Floreat Lucina."

The President, in accepting the chair in the name of the Society, said:—

Gentlemen, it is at once a pleasure and privilege to me to accept, on behalf of this Society, this handsome gift from one of our most honoured members. I congratulate the Society, and I thank Dr Church most cordially for his kindness and generosity.

The letter which you have just heard read expresses in very warm terms how highly Dr Church estimates the work of this Society, and we value his appreciation, as we recognise in him one of our most cultured and esteemed Fellows. His interest in our Society has culminated in the gift of this beautifully chased and handsome President's chair, which is to grace our new hall.

This is the first President's chair that the Society has ever possessed, and for the beautiful and gracious words of his letter, as well as for the gift itself, I beg to offer Dr Church our profound and most sincere thanks.

This chair is to accommodate for all time coming him who has the honour of presiding over this Society, and I trust that

in the years to come, it may be filled by one who will worthily follow in the footsteps of its distinguished founder.

We most gladly and gratefully accept this handsome gift from one of the most learned members of our Society, namely, Dr Henry Macdonald Church, and while thus thanking Dr Church, I shall ask the senior Secretary to send him a permanent acknowledgment of his kindness.

Gentlemen, I am very proud indeed to be the first occupant of this chair, and the only posthumous honour I can ever hope for, will be to be handed down to posterity as "Père la Chaise."

Dr Church replied:—Mr President and Fellows, I have to thank you most cordially for the honour you have done me in accepting this gift. The only justification I have in offering the gift is because I am one of the senior Fellows. It was very nearly forty years ago, in 1875, that I became a Fellow of the Obstetrical Society of Edinburgh, when Dr Matthews Duncan was in the Chair, and Dr Charles Underhill was Secretary of the Society. I have been much impressed in looking back and realising what an advantage it has been to me in my professional life to be associated with the Obstetrical Society. I hope that all the young members of the profession, both in town and country, may become members of this Society and have the benefit I have had.

When one thinks of the papers that have been read, of the discussions which have taken place, and of the presidential addresses, one feels what a valuable experience and privilege the Fellowship has bestowed. Dr Underhill while Secretary became in 1888 President of this Society, and I don't think there is anything more impressive, or reads more like a romance, than the history of the Obstetrical Society from the beginning up to that date; it would well repay anyone to read Dr Underhill's paper in the *Transactions* of 1888.

I would just refer to one point which has interested me, and

that is the motto or crest of this Society. I do not know, nor have I been able to find out, in whose classical brain this motto had its birth, but it is an interesting motto and deserves thought and a little study. The artist fortunately has done the carving on the back of the chair very well; it is an exact copy of what one sees on the *Transactions* of the Society. In the first place, one notices the beautiful female figure, which is supposed to be Juno or Lucina, the old mythological goddess who presided over marriage and childbirth; women who wanted an easy labour made obeisance to this Goddess Lucina, *Floriat Lucina*, let Lucina flourish. Lucina and Juno, the goddess of heaven, are one and the same. Between the motto and the figure are two beautiful olive branches fertile with young olives.

I think it is auspicious that we have you, Sir, as President of the Society, because if Lucina or Juno, the mythological Queen of Heaven, may have helped these poor women in their distress, it must have been merely by an act of faith on the part of the suppliant. Although Lucina did a great deal indirectly to relieve these poor women, have not you done infinitely more through the use of *Scopolamine morphine narcosis* in making labour easy and pleasant without at all injuring the child? The Queen of Heaven must claim you as a distinguished son.

If am afraid the chair has been got up on somewhat Spartan principles, but I hope it may be comfortable although not luxurious. I feel very much the kindness the Society has shown in receiving from my hands this gift. I thank you cordially.

II. *Dr Keppie Paterson* showed—MEMBRANOUS CAST OF UTERUS, interesting because of its rarity. The patient, æt. 40, suffered from severe pain. A peri-rectal cellulitis was diagnosed; this was treated and cleared up, but intense dysmenorrhœa continued and the patient said she was passing

clots and pieces of flesh. What was thought to be a piece of membrane was detected. Then one day the patient brought this specimen. The patient was an unobservant woman and it was difficult to get a definite history. She had had one child about eleven years previously, but had suffered from dysmenorrhœa before that and severe pain since. The casts were passed very often and, so far as could be ascertained, she had severe dysmenorrhœa every time. Described like labour pains. She said she had had no miscarriages, but had passed two periods six years ago; the trouble had, however, begun before that. The periods were now quite regular, although perhaps more frequent than usual; there was no menorrhagia, but some leucorrhœa.

Dr Johnstone had kindly examined the specimen, and reported that it was a solid triangular cast of the cavity of the uterine body. Microscopically it consisted of a fibrinous network with masses of leucocytes, and on the outer surfaces portions of columnar epithelium from the surface and superficial glands of the endometrium.

III. *Mr N. T. Brewis* showed—(*a*) VULVA REMOVED FOR EPITHELIOMA ASSOCIATED WITH LEUCOPLAKIA. The patient, an unmarried lady, æt. 67, for two years prior to being seen by Dr Brewis had suffered from vulvar itching and irritation. The specimen illustrated the most important sequel of leucoplakia. In the right labium minus and clitoris an epithelioma was to be seen, while leucoplakia extended over the vulva and perineum. The inguinal glands were not involved, and as the excision was made with a wide margin of healthy tissue, the prognosis was good.

(*b*) LARGE FIBROID TUMOUR GROWING FROM THE FUNDUS BY A THIN PEDICLE WHICH HAD BECOME TWISTED AND STRANGULATED, removed from a patient æt. 47. The tumour was universally adherent and parasitic. On section well-marked

necrobiosis was seen. This was by far the best example of a twisted pedicle of a fibroid tumour he had ever seen.

(*c*) UTERUS AND BILATERAL SOLID MALIGNANT OVARIAN TUMOURS, removed from a patient æt. 50, a single, nulliparous woman. Both ovaries were non-adherent, were hard in consistence, and microscopically masses of epithelial cells were seen surrounded by fibrous stroma. Scirrhous cancer of the ovary was nearly always bilateral, and practically always metastatic, though the primary focus may be undiscoverable at the time of the operation. In the present instance, no evidence of malignant disease elsewhere was found. It was now five months since the operation, and as the patient was in excellent health, it was hoped that this case would prove an exception to the rule.

(*d*) BODY OF THE UTERUS ALONG WITH FIBROMA OF EACH OVARY. The patient, æt. 64, the mother of six children, was sent to Dr Brewis by her medical attendant on account of hæmorrhage and prolapse of the uterus. On careful examination it was found that the perineum was deficient, the vaginal portion of the cervix was greatly hypertrophied, and there was a well-marked cystocele and rectocele. In the abdomen a hard round swelling was felt. The uterus was curetted, and a small suspicious-looking portion of the cervix excised, and, along with the scrapings, submitted to microscopic examination. There was no evidence of malignancy in either. On opening the abdomen both ovaries were found greatly enlarged, forming the tumours now seen. The tumours were removed along with the body of the uterus and the cervix stitched into the abdominal wound, with the object of curing the cystocele. The abdomen was then closed and the rectocele operated on. Fibromata of the ovary were nearly always unilateral.

(*e*) UTERUS REMOVED FOR PYOMETRA. The uterus was seldom removed for pyometra unless it was associated with cancer, as was usually the case.

The patient, æt. 63, had had eleven children. For a year or more she had been troubled with a copious purulent discharge from the interior of the uterus. The uterus was enlarged, sound passing 4½ ins., retroflexed, very soft in consistence except at the fundus, where a hard body was felt. Curettage and irrigation continued for a long period did not in any way check the discharge. The pus contained staphylococci and the uterine tissue was so extremely soft that it was considered even curetting was dangerous. Hysterectomy was, under the circumstances, thought to be indicated, and the uterus was removed by the vaginal route. On laying the uterus open a small fibroid was seen in the fundus. There was a distinct uterine cavity, and the flexion imprisoned the secretion, which had become septic.

IV. STILL-BIRTHS' REGISTRATION.

(Adjourned Discussion.)

By J. W. Ballantyne, M.D., F.R.C.P.

Dr J. W. Ballantyne read a paper on still-births' registration at the Meeting of the Society in March. The subject was referred to the Council for consideration and report. The Council recommended the following modifications of Dr Ballantyne's conclusions:—

1. That, in view of the existing confusion in the terminology of still-births and allied states, an attempt to reach uniformity of definition should be made.

2. That an Ante-natal Deaths ("Still-births") Registration Act is greatly needed in this country.

3. That the definitions of dead-birth, live-birth, still-birth, and abortion should be so constructed as to be in accordance with the physiology of the time of life dealt with. The following definitions are suggested:—

(*a*) For *dead-birth*—the complete expulsion from the

maternal birth-canals of a child which, during or before birth, has lost the characters of ante-natal life (heart-beat, arterial pulsation, and movement).

(*b*) For *live-birth* — the complete expulsion from the maternal birth-canals of a child which, whilst it loses pulsation in the cord, adds on to the other characters of ante-natal life the special sign of post-natal vitality—viz., pulmonary respiration.

(*c*) For *still-birth* — the complete expulsion from the maternal birth-canals of a child which, whilst continuing to exhibit one or more of the signs of ante-natal life (heart-beat, arterial pulsation, movement), fails for a time to assume that of post-natal life (pulmonary respiration), and then either loses even the characters of ante-natal life, or is successfully resuscitated (transanimated), and assumes those of post-natal existence.

(*d*) For *abortion* or miscarriage—the termination of ante-natal life (by the expulsion of the uterine contents) before the end of the sixth month. A *premature birth* is expulsion of the uterine contents after the sixth month, but before the full term; and it may be a dead-birth, a live-birth, or a still-birth.

In reopening the discussion, *Dr J. W. Ballantyne* said:—Mr President *in cathedra!*—At your request I shall try to say something in a very few words to link on the discussion of to-night with the reading of my paper some weeks ago.

My paper was really a consideration of terminology, but the terms discussed had not merely an academic but a practical importance and bearing. There was no difficulty with "registration" and "notification," but with the terms "still-birth" and "dead-birth," and with the equivalents of these terms in foreign languages, there was not only extreme difficulty, but also immense confusion. The great problem standing in the way of all attempts at definition was the fact that in many cases one had to wait a little while before

being sure that any given birth was going to be a live-birth or not. Was the child going to be still or going to move, or was it so stilled before birth as to be for ever beyond the possibility of moving again?

My explanation of the confusion was (1) a lack of adequate recognition of the physiology of ante-natal life, and of the changes which occur or may occur at birth (the transition time between ante-natal and post-natal life); and (2) the habit which the public, with the connivance of the profession and the profession itself in some places, had got into of regarding still-birth as a synonym of dead-birth. I have it from Dr Amand Routh that a still-born baby is dead and cannot be resuscitated, and yet in obstetrical text-books and in the *Journal of Obstetrics* itself we read constantly of the treatment of still-births and even of the best methods of resuscitating still-born babies! It was common to hear a medical man say, "I was not sure whether it was still-born or dead."

Consequently as Parliament was soon to bring in legislation dealing with "still-births" and their registration, it seemed to me well to discuss the terminology of the subject before the words still-birth, dead-birth, live-birth, etc., were finally crystallised along lines which are erroneous. I drew up some definitions, and these with some modifications which I regard as improvements had been unanimously adopted by the Council of the Society.

Dr W. Leslie Mackenzie (Local Government Board) thanked the Society for permitting him to be present at a discussion which was so entirely outside his usual work. He had read Dr Ballantyne's paper with the greatest interest, and doubted if he could contribute anything of profit to the discussion. Assuming that these differences exist, the question was, how can they be given effect to in practice? First, the

Notification of Births Act (1907), which Dr Ballantyne referred to, simply speaks of notifying the birth of a child which, as Dr Ballantyne says, includes every kind of birth. No doubt that will be of practical value to Medical Officers of Public Health, as giving a hint to further notification. Dr Chalmers of Glasgow was finding it of the greatest value in one respect; he has supplemented it with the free use of the Wassermann reaction. It was important not only in this connection, but also in infectious diseases as well. Secondly, in Scotland, although the Act has not been adopted under pressure to any great extent, the actual population under notification is rather more than 50 per cent.; that is, nearly two millions and a half people are under notification. In connection with the Dewar Committee for the Highlands, it was suggested that the notification of births should be made in the Highlands. One felt that three things hung together, namely, the notification of births, the proper regulation of midwives, and the whole question of infant mortality. These three Acts hung together, and when the notification of births became universal it would undoubtedly help. The Local Government Board had practically determined, where the Act was not adopted, to have it done by themselves. This Act, on the whole, in spite of the unfortunate exception that the medical profession were not paid for the duties required of them, had been fairly satisfactory.

In connection with the Midwives Act, one line in which the use of it might be extended was in providing training institutions. The local authorities were asked to contribute for the training of midwives. The training of midwives under the Board should include definite and special teaching on still-births on the pathological lines that were considered obviously desirable.

The one question he had practical doubt about was, and he was glad that it did not fall to his department to settle,

whether, allowing that these four distinctions were desirable for other reasons than scientific precision, and he thought that Dr Ballantyne's paper justified that, it was a practical question. Were they not in danger in accepting still-births according to Dr Ballantyne's definition—"The complete expulsion from the maternal birth-canals of a child which, whilst continuing to exhibit one or more of the signs of ante-natal life (heart-beat, arterial pulsation, movement), fails for a time to assume those of post-natal life (pulmonary respiration, crying, etc.), and then either loses even the characters of ante-natal life or is successfully resuscitated (transanimated), and assumes those of post-natal existence"—seeing that it has got such a deep-rooted association with a dead-born child, of confusing rather than simplifying? Personally he would risk the confusion and raise the standard of the general practitioner's knowledge. A vast amount of statistics could be got even although some considered a dead-birth meant one thing, a live-birth another thing, and a still-birth a third thing; while others classed them all together. After reading Dr Ballantyne's paper he believed that even a limited amount of statistics in the hands of good men like the local authorities of Glasgow and Edinburgh and other large centres would be of great value. If the Registrar-General had to distinguish five forms of tuberculosis and two or three forms of pneumonia, it was not asking very much to ask the physicians throughout the country to make these distinctions as regards births. In a pamphlet by Miss Margaret Bonsfield of the National Women's Guild, which was to be discussed in London the next day (14th May), stress was laid on this point. She said, "Compulsory notification of births is a necessary part of our scheme, and our attention to still-births is urgently needed." This pamphlet was based on a very large experience of different public bodies, and the writer came to the same conclusions as Dr Newsholme, who, in connection with the Venereal

Commission, was equally strong on the notification and registration of still-births.

Dr J. C. Dunlop said, like Dr Leslie Mackenzie, he had read Dr Ballantyne's paper with the very greatest interest. Up to a certain point he was absolutely at one with him. He quite agreed more accurate definition was required, and he quite agreed that interesting and important information might be got, but he disagreed as to the method of getting it. He disagreed about the use of registration for the purpose instead of notification. Let them consider what registration was. Registration was not an administrative machinery for the production of vital statistics; vital statistics were a by-product, they were of great value and interest, but they were not the primary function of registration. Registration was producing a record of civil rights, and these records were preserved and kept for all time. Now was that what was wanted with dead-births? Fifty years hence would it be of any interest to anyone to know that a father or a mother had a dead baby? It did not fit in to the ordinary principles of registration. Undoubtedly registration would produce excellent statistics, but that might be got equally well by compulsory notification. One must look at the practical aspect. Further, registration was done by registrars, who in Scotland were a very intelligent set of men, but they had no medical knowledge; they would not know whether a child had lived a few hours, or the duration of pregnancy; they could not examine the mothers and ask them when they last menstruated; the mothers did not know the length of pregnancy, how then could the registrar discover whether it had gone on for six months? How were they to know whether the cord pulsated or not? There was no medical supervision over registration until the matter went to his room in the Register House, where the use of the material was restricted to the production of statistics. The absence of

medical supervision was a very important argument against the registration of these cases compared with notification. His first point was that one did not want a public record, and the second was the absence of medical supervision. The registration of these cases did not fit in with the established system of registration in Scotland. They had three registers, namely, those of marriages, of deaths, and of births. The birth register only dealt with children who live, in order that these people when they grew up might have their civil rights—old age pensions it might be. A still-birth or a dead-birth did not fit in. Dr Ballantyne recognised this, and advised no fewer than three additional registers—one for miscarriages, one for dead-births, and one for still-births. Such a scheme was very complicated, and one feared the ordinary system of registration would be upset. Such duties were beyond the capabilities of an ordinary registrar.

One important point—Dr Ballantyne suggested that all still-births, that was, the births of children who were capable of reanimation, should be registered in the still-births' register; if they survived they should remain there, but if they died they should pass on to the death register. He (Dr Dunlop) was entirely opposed to that—if the child died let it remain on the still-birth register, and if it lived, go on to the live register.

The principal object of notification was to supply information in order that the Medical Officer of Health might take action and supervise the cases. (1) The Medical Officer of Health should be directly notified, and (2) it should be done in secret. Imagine an unfortunate girl who had an illegitimate miscarriage! If the information was given to the Medical Officer of Health it was in confidence—the confidentiality was *the* thing. (3) The knowledge was passed on to a man with the technical knowledge necessary to appreciate it. And, lastly, notification to the Medical Officer in no way complicated the present good system of national registration, a system, let it

be remembered, which dealt with established civil rights. Further, was there any hope whatever of getting completeness in this matter? The whole movement was in order to make the birth knowledge more complete than it was at present. Could they expect even compulsory notification of still-births and dead-births, including miscarriages, when they frequently were not recognised, and when the whole tendency was to hide them? Completeness could not be got without the notification of pregnancy itself, and that was too great an interference with the liberty of the subject and with ordinary privacy to be recommended or even considered here or elsewhere.

Professor Littlejohn said that before going on to discuss the paper, he wished to say a word or two with regard to the remarks Dr Dunlop had made. He spoke with all humility in the presence of the medical member of the Local Government Board of Scotland, but the chief function of statistics was not purely civil. The whole progress in public health since registration was introduced had been in the direction of preventive medicine. He appealed to Dr Leslie Mackenzie to say whether the registration of vital statistics had not been of the greatest importance in combating disease in various forms. He denied that Dr Dunlop was right in assuming that the chief function of registration was to register births for civil purposes; it was equally in the interest of public health, and nothing more valuable had been done in the cause of preventive medicine. If anything was to be gained by registration of still-births in the interest of public health, apart from the *notification* of dead-born children, then by all means let it be done. Dr Dunlop stated that in the absence of medical supervision registration was valueless. Yet he accepted the death certificates of unqualified persons—a medical student it may be—classified them, and used these statistics. Why not take a midwife's certificate for what it was worth?

With regard to the paper itself, he did not wish to be purely destructive, and thought it was always making progress when a difficult question like this was discussed. But could not Dr Ballantyne's definitions be simplified? What did they want to get at? They wanted to know how many viable fœtuses were dead-born. Let there be a notification of dead-born children, that is, children which might have died some weeks before or have died during birth. One did not require any further subdivision. They wanted to know whether a child died of constitutional disease (syphilis) or from some accident. They wished information as to whether a child died from the cord being round its neck, or from some other cause during birth, or from some constitutional condition. Dr Ballantyne said (on page 219):—"At the same time, there are good reasons for trying to make the distinction between ante-natal and intra-natal deaths, and the absence of macerative changes in the latter makes the separation possible." There could be no such separation possible if the child died within two or three days of birth. It was only when there was maceration one could tell, and a fœtus dying *in utero* showed no macerative changes for several days. With reference to still-births, which were really live-born children—children born living—who gave some sign of life although respiration was absent. Dr Ballantyne bound himself to the test of respiration. That was the criminal law; according to civil law, movement however slight of a limb or an eyelid was sufficient to designate live-birth. The still-born child was one which was born without giving any obvious sign of life; but the heart was still beating, only the respiration was wanting, it was born in a state of asphyxia; they were easily revived, and came to life—trans-animated Dr Ballantyne termed it. What was to be gained by knowing that a child had been silent-born. A thousand and one causes might give rise to it. But if it became live-born by some little handicraft on the part of the midwife

that child was legally born alive. Therefore what was to be gained by registering or knowing that a child had been still-born? Unless one wanted to accept the view that, as some doctors meant by still-birth, it was a dead-born child. There were dead-born children and live-born children, and he did not think anything was to be gained by further classification.

Dr Berry Hart thought they were indebted to Dr Ballantyne for bringing up such an important subject. There was no doubt that the question of statistics of all kinds, especially vital statistics, or in this case dead statistics, were of the highest importance. That was shown many, many years ago by the eminent observer Quetelet, whose book is most fascinating reading, and shows how much one can learn from the study of probabilities.

Now, the object of registration is to gain information as to the health of the nation. He did not agree with Dr Dunlop, who would limit the duties of registrars to recording the names of those born or who had died. What lay at the bottom of still-births and abortions was really the national health. They wanted to find out the influence of syphilis in producing premature labours and dead-births, they wanted to find out, too, the influence of rickets in producing dead infants from narrowing of the pelvis. It would be of great importance if it could be shown that in time there was a diminution of dead-births and abortions, for it would show that the national health was improving and that syphilis and rickets were becoming less. The great point is to get facts. That was the great difficulty he had in writing his paper on Insemination-Labour Curves. He had suggested to the Insurance Committee that they should give cards which the doctors should fill in, giving the date of labour and the data of the calculation. His strong opinion was that one should try and get statistics

in such a way and, of course, as judiciously as possible. The registrars could thus collect most useful observations.

It was necessary, however, to be very judicious in what one asks; still-births, dead-births, and live-births was as much as one would get, and personally he was a little doubtful about the still-births. Professor Littlejohn, he thought, was quite right in pointing out that it was a refinement. It was a verbal thing, and open to the criticism that the child was still-born but lived for a few minutes; in that case the child, he thought, should be registered as dead-born; but if a child is still-born and lives for some days, it should be registered as a live-born child, and then afterwards as dead. As many statistics as possible should be obtained, but they should not ask for still-births, but for live-born and dead-born. As to abortions, he thought the statistics would be absolutely worthless if they were asked for.

Dr Dunlop.—The point was that registration was for civil purposes; the records were in constant use. Vital statistics were only one thing.

Dr Berry Hart.—Dr Dunlop deprecates registration. Personally he held that this registration was of the utmost value as giving information of the condition of the nation at a certain time, so that one could compare it with that years afterwards.

The President.—It is quite clear. One could register in the books under the Registrar-General. Notification was a purely public health matter affecting the Local Government Board.

Dr Keppie Paterson said he admired the amount of work in Dr Ballantyne's paper; it meant an enormous amount of collaboration and research to produce such a complete paper. After hearing Dr Dunlop he thought it would be wise to call it

"Still-births' Notification." Undoubtedly it would be far better to go through the hands of the Medical Officer of Health than through those of the Registrar. Notification should be made compulsory all over the country, and it must be secret between the doctor and the Medical Officer of Health.

With regard to the scheme itself, Dr Ballantyne had defined an ideal scheme to obtain a wider knowledge for the sake of national eugenics. The definition of still-birth is ideal, but some difficulty might arise in the notification. The problem is how it is to be carried out. If it is to be compulsory, then some of it will require to be done by trained midwives. It might be tried, but if even not quite successful, it was always a step on in the proper direction. In lying-in institutions, in the maternity hospital or in dispensary practice, where one can deal easily with a patient, one might get important information to send to the Medical Officer of Health; but in private practice it might cause a great deal of trouble if the cause of the still-birth or dead-birth was hereditary. A great many still-births occurring in a lady are, of course, due to syphilis. That is equally realised by the Commission on Venereal Diseases, and the majority of those giving evidence is against the compulsory notification of syphilis and venereal diseases. Well, now, if a doctor was compelled to put in the cause of a still-birth or dead-birth as syphilis, it might involve him in a great deal of trouble in his practice, because in most cases the husband denies it straight. What was one, then, to do? What was to be done to find out what the cause is? The husband will not submit to a Wassermann test, and what was to be done? And if the cause of dead-births and still-births is not to be put in, of how much value are these statistics? It would always be a step in the right direction, but it would involve a very serious difficulty. Many medical men would never enter the cause at all if demanded in the certificate, or would insert in such a way as to be useless.

Dr A. H. F. Barbour also regretted he was not present when the paper was read; he had only glanced over it very hurriedly. On looking over the definitions, the first thing that occurred to him was with regard to abortion or miscarriage. This was described as "the termination of ante-natal life (by the expulsion of the uterine contents) before the end of the sixth month." Now, there is no evidence of ante-natal life until four and a half months. During the first two or three months there is no evidence of ante-natal life. He quite agreed that it would be well to have abortions registered so as to show the tendency to diminution of abortion; he did not think, however, they would get any statistics, because it would be necessary to show that these abortions occurred in the same family, and also that the frequency of abortion showed that it was related to infection.

Then premature birth, "expulsion of the uterine contents after the sixth month, but before the full term, and it may be a dead-birth, a live-birth, or a still-birth." He was not sure that it would not be better to extend that. That was the practical question, whether the child was alive or not. He was not sure whether it would not be better to say "expelled at the sixth month"; whether one should not extend that to the birth of a viable child. Looking at the question practically it resolves itself into this—What one wants to get at is the health of the community. In order to do that, two classes of cases must be distinguished, namely, the cases in which the child dies *in utero* and is expelled subsequently, because the uterine wall will not retain a dead body, at what time it takes place did not matter; and the death of the child during the process of labour. In dealing with those cases the subject becomes complicated. In the first case there is some constitutional cause affecting the health of the community, while, in the second, one is at once brought up against the bad management of the labour on the part of the midwife or medical man. The

statistics of death during actual labour may have a bearing on the health of the community; he thought that in a large proportion of cases the management was improving, there was now a better class of midwives. The point he wanted to emphasise was, if, in the first case, one could prove that the child died during labour, then one was brought up with a number of other causes which had nothing to do with the community. Prof. Littlejohn remarked that they had no knowledge as to whether the child died before birth. Personally he drew a hard-and-fast line between the cases of death of the child *in utero* and expulsion afterwards, and death of the child during the process of labour.

Prof. Littlejohn.—Dr Ballantyne says maceration.

Dr Barbour.—One goes back to an earlier period. The important point is to get information as to whether the child died in labour or not.

Dr James Ritchie regretted that he too had not heard the paper read. He admired very much the great amount of labour and the interesting character of Dr Ballantyne's paper. He (Dr Ballantyne) advocated the compulsory notification of births after twenty-eight weeks' gestation. For scientific purposes it would be desirable to have compulsory notification of all pregnancies and abortions, but he did not think it was possible, they would be so incomplete as to be really impracticable. If the compulsory notification of all births after twenty-eight weeks of gestation could be got, they would be sent to the Medical Officer of Health, who would collect the statistics that were described, and then they would be forwarded to Dr Leslie Mackenzie and become valuable. He quite agreed with Dr Berry Hart that the more vital statistics that could be got the better. He believed really valuable

statistics would be returned by the medical men or midwives; they would be paid for it, and should be asked to furnish certain statistics, a form of which might be drawn up. He doubted Dr Keppie Paterson's statement as to why doctors could not give these returns.

As Dr Ballantyne pointed out, whether death took place before or during parturition, it was of great importance that the statistics should state on what day death occurred and, so far as is known, what was the cause of it.

Dr Haig Ferguson wished also to add his testimony of appreciation of Dr Ballantyne's paper, which showed a great amount of study and investigation.

The discussion brought out that notification of still-births was important, and he was much struck with what Dr Craufurd Dunlop had said as regards the difference between notification and registration.

From a scientific point of view Dr Ballantyne's paper was all that could be desired, but from a practical and legislative point of view it seemed to him to be far too elaborate for useful purposes. The question, of course, arises as to what still-birth really is. From the popular aspect a still-birth means a dead-birth, and this one saw in the newspapers every day. This view of the question could not easily be got rid of, and it seemed to him that for legislative purposes one ought to adhere to this popular belief.

It would be more useful to define what we *medically* mean as a *still-birth* as "Asphyxia Neonatorum," and use the term "still-birth" as it is used in the popular sense, to mean a "dead-birth."

The Ceylon Government's definition that "still-birth means a child born after the 28th week of gestation as dead, or apparently dead and not called back to life," seems very practical and clear.

With regard to Dr Barbour's remarks as to distinguishing a child dying before birth from a child dying during labour, he (Dr Ferguson) must protest from what Dr Barbour said. The question of the mother feeling or not feeling life shortly before the birth is of no value whatever. He had known women who had felt no fœtal movements bearing living children, and other women who averred they felt fœtal movements who were not pregnant at all. Personally he would put no weight whatever on the subjective phenomena of fœtal movements as an indication of fœtal life, apart from other manifestations.

He had listened to the discussion with great interest.

Dr J. W. Ballantyne said it was no light business to reply to all the questions which had been put.

With regard to Dr Leslie Mackenzie's remarks: his difficulty was whether such an Act could be made practical enough to be of any real value. Was not this just the history of all registration and notification? What about the difficulties which were suggested when tuberculosis first began to be registered; the same with infectious diseases? There has always been great difficulty in introducing these things, but the medical profession with better knowledge and training has always in time been able to accomplish the end sought. They all knew there were medical Acts of Parliament being passed now, but in less than ten years there would be other ones to rectify or add something to them. He agreed strongly with Dr Leslie Mackenzie that any slight risk ought to be faced, because he believed something valuable would come out of such an Act as the still-births one. While in London, he had spent an evening talking over the subject with Dr Newsholme, whose idea was that in registration or notification schedules the "causes of still-birth" should be put down with the words "if known" appended, and that after, say ten years, when the

profession had become accustomed to this, the words "if known" could be omitted. Of course, he said, most of the medical men would at first put down nothing, they would simply draw their pens through it, but a few men would answer, using the terms "probably so-and-so" and "presumably so-and-so," and by-and-by with increasing knowledge the "probably's" and "presumably's" would be left out.

Then as to Dr Craufurd Dunlop's remarks: he (Dr Ballantyne) found the whole body of hygienists discussing legislation about still-birth registration, and it never occurred to him that notification was a serious alternative. He could see there were difficulties, but he thought Dr Dunlop must discuss them with the other Health and Statistical Authorities. Personally he thought it would be quite possible to make a good beginning with compulsory notification. Could Dr Dunlop say if his idea was new, or if other vital statisticians were of the same opinion?

Dr Dunlop.—I have good reason to believe that there is a very considerable official opinion against the registration of still-births.

Dr Ballantyne.—So that if the Bill comes in within the next few years it will be a Still-births Notification Act?

Dr Dunlop.—Certainly a Still-births Registration Act would be objected to by many.

Dr Keppie Paterson.—The notification is secret.

Dr Leslie Mackenzie.—It is a controlled affair.

Dr Ballantyne, continuing.—With Prof. Littlejohn he quite agreed; the matter should be simplified. He was horrified

when he saw the length of his own paper! But was there not a danger of simplifying it too much? In connection with the third group—that of the still-births—he did not remember that he had asked that they be separately notified. These were not the things he wanted specially registered; what was needed, was to find out what was the number of ante-natal deaths, and how many babies were born still but capable of being resuscitated. If they recognised that there was a distinct difference between a still-born baby and a dead-born baby, were they to "fall in" with a popular and incorrect nomenclature, simply because people did not like to have their names connected with a dead-birth in the newspapers, and liked to put still-birth instead? They should retain the word "still-birth" for certain cases. He thought it was a beautiful word, if one might use the term in this connection; for the child literally lay *still* until one began to see the quiver over the epigastrium which betokened the first sign of post-natal life.

The President.—That is a live child to all intents and purposes.

Dr Ballantyne, continuing.—Take the quiver of the eyelid to which Prof. Littlejohn referred as a sign of post-natal life. They knew that was not a peculiarity of post-natal life only, but of ante-natal life also, for there was every reason for thinking that before birth the child has been opening its eyes as well as kicking about. One could open one's eyes under water! He did not think that was such a good means of defining post-natal life as respiration was. The word respiration was introduced into the definitions as a modification by the Society's Council in order to get simplification. He had stated the characters of ante-natal life as heart-beat, arterial pulsation, and movement; but the findings of the Council were that pulmonary respiration was better, in order to simplify the definition. He also agreed with Prof. Littlejohn and would be

content with the two classes for registration purposes, born dead and born alive, but he thought there would be some difficulty, because of the child being born alive in one case in an ante-natal sense and in another in a post-natal. It was really necessary to attempt to distinguish between the two kinds of life. He did not think that Prof. Littlejohn had thought out all the subtle differences between life before birth and life after birth. To put it simply, the infant is a fish before birth, and a mammal after; the placental respiration before birth is entirely different from the pulmonary respiration after birth. That was the explanation too of his term "transanimate"; one did not return the child to the same kind of life, one carried it over to another type of life, made it live in a new sense.

Prof. Littlejohn.—Use life in the popular sense.

Dr Ballantyne.—But this is the chief test in the difference between post-natal life and ante-natal life. The one great change is that the blood instead of going to the placenta goes to the lungs. He quite admitted the importance of Prof. Littlejohn's criticism; it certainly was desirable to get the easiest observable sign of life for the general practitioner to take note of.

With Dr Berry Hart he quite agreed that this was a national health business, and that it is for the future and the health of the nation that they were striving in this matter, not so much for the immediate value of the statistics as for the future. Infantile diarrhœa has been largely controlled and some other diseases which were causes of death in children, but very little was known of the causes of death which the child brought with it into the world but which did not become operative until after birth. In Paris they were now up against the causes of death which were present before birth but only became evident after birth, such as the various malformations and diseases of the

fœtus, and which did not interfere with life and health before birth. He did not include the children who were dead-born because of malformations, but spoke of those who died two or three months afterwards from what were called congenital or ante-natal causes.

Dr Wm. Fordyce.—Give one.

Dr Ballantyne.—The various heart conditions, for instance, giving rise to "blue babies." According to the New York statistics, something like 33 per cent. of the infantile deaths were due to those things which come into evidence after birth but were present before it. Now they had to try to treat these ante-natal things; that was why they wanted to know about the things dangerous to the child before birth, the dangers which arose in its birth, and the things which were not dangerous before or in the birth but which began to be so after its birth. He believed the only way to get at them was by registration or notification of ante-natal deaths such as he had indicated. They could not diminish much more the mortality after birth along present lines, something more had to be done along new lines; and they had now to tackle the second big block of causes of infantile death, viz., the congenital ones.

Dr Keppie Paterson had said a little about the enormous amount of work he (Dr Ballantyne) had put into the subject; but most of the statistics, it was right to say, were got from the very valuable report of the Committee on Infantile Mortality in the *Journal of the Royal Statistical Society;* it was there he had got information as to definitions in use abroad, etc. The differences of opinion would never have been known had it not been for this inquiry carried out in London. For instance, the French used the term *mort-nés.* In France a child was called *mort-nés* if dead-born; but two or three days were allowed before registration takes place; if the child was

born alive but died before the second or third day, it was still called *dits mort-nés.* This was very confusing, and it meant that all French statistics were useless for comparison for those of other countries in this matter. They have been trying to simplify it, but instead they seem to have made it more complicated.

Dr Barbour had referred to the age of the pregnancy. His (Dr Ballantyne's) chief point really in raising the question of miscarriage and abortion (he was not at all hopeful of getting registration of early miscarriages) was because he thought it well to speak about it and define it, for most people have not gathered that a miscarriage was *a death,* an ante-natal death. He had told the incident before of the lady who came to him and said that since last he had attended her in confinement she had had two miscarriages. When asked if she had any doctor for them, she replied, "No, they came away." "Why are you coming to me now?" She replied, "Well, I have learned something lately. I was looking at the other children at the table the other night, and I thought if these two miscarriages had not occurred perhaps there would have been two more, and I began then to realise that there was some meaning in a miscarriage." "Oh, yes, you did not realise (he had said) that these were family deaths, but that there was no following funeral?" She said, "That was just it." Her next question was, "How can these be prevented?" That incident showed how little people realised that an abortion was the death of a fœtus. Dr Barbour's objection, therefore, largely fell to the ground. All that he (Dr Ballantyne) wanted was to have these things in some way taken notice of. The fœtus was there; it must have been alive or else it could not have been 2 or 3 ins. in length. Dr Barbour has spoken of the term viability; this was a term which he (Dr Ballantyne) had carefully avoided, because it was one which gave rise to the greatest difficulty between doctors and lawyers.

Professor Littlejohn.—In what way?

Dr Ballantyne.—Well, what would you term viability?

Professor Littlejohn.—A child which is alive.

Dr Ballantyne.—Well, sir, I attended your honoured father's class, and he indicated there were difficulties about the date of viability.

Professor Littlejohn.—You asked me, sir, not my father.

Dr Ballantyne.—I appeal to you, sir, as the son of your father. Doctors were sometimes asked by lawyers, What is the earliest date of viability? He wanted to fix the date of viability. Was it not true that the date of viability was being pushed a little farther back in ante-natal life by new means of treating premature infants.

Professor Littlejohn.—Give an instance where the question of viability is of any practical importance.

Dr Ballantyne.—By the invention of a better class of incubator the fœtus has been made capable of survival at an earlier ante-natal age.

The President.—Of premature labours 35 per cent. die.

Dr Ballantyne said the date of viability could not be fixed. They now could keep a child alive earlier; it was not a question of age so much, but of the health of the parents. A child born at the eighth month of syphilitic parents was not nearly so likely to survive as a child of six and a half months with healthy parents.

Professor Littlejohn.—Viability is at seven months.

Dr Ballantyne.—About twenty-eight weeks. He had introduced the words twenty-four weeks into his definition because of his talk with Dr Newsholme and Dr Stevenson; he had gathered that there was a desire to push this date farther back so as to catch more cases in the notification net. He thought the new incubators, "thermogenic nurses," and the like took the date of viability back to the sixth month (24th week).

Dr Haig Ferguson's criticisms had been answered already. He (Dr Ballantyne) asked, How can you make a complicated business simple? Was it possible to make such a complicated thing as this still-births simple without neglecting some of the facts? Thus the term "called back to life" was inadequate. One did not call the fœtus back to life, one transanimated it from the fœtal to the infantile type of life.

Meeting VII.—June 10, 1914.

Sir Halliday Croom, *President, in the Chair.*

I. *Dr M'Master* showed—two specimens of hydatidiform mole smaller than a normal ovum of the same age, which, he pointed out, went to prove Dr Briggs' contention that in the majority of cases the mole was smaller than an ovum of the same size.

There was some discussion upon the specimens, and Dr M'Master was asked to read a paper on the specimens so that there might be opportunity for a true discussion.

II. NOTES ON A CASE OF PREGNANCY IN A MALFORMED UTERUS.

By William Fordyce, M.D., F.R.C.P.

Mr President and Gentlemen,—There is a large literature on the subject of pregnancy in malformed uteri, but the following case which recently came under my care presents some points of special interest which make it, I think, worth recording.

On 20th December last I was asked to see a young married woman, æt. 20, by Dr Hope, Comely Bank. The history of the case is as follows:—She had been married for six months, and for ten weeks previous to my seeing her she had suffered excessively from vomiting in connection with pregnancy, which was supposed to be about three months advanced. She also complained of pain in the right side of the pelvis, and on one occasion about one week prior to my seeing her there had been a slight discharge of blood from the vagina. Dr Hope, on vaginal examination, had detected a soft swelling beside the

uterus, and thought the case might be one of extra-uterine gestation.

The cervix was soft and the external os was normal. The swelling beside the uterus was easily made out, and bulged down into the right lateral and posterior fornices, and was closely connected with what seemed to be a small firm uterus pushed over to the left side. The upper border of the swelling could also be palpated quite distinctly through the abdominal wall in the right iliac region, and was distinctly tender. At first I agreed with this diagnosis, and advised the removal of the patient to the Royal Infirmary, where she was admitted about one week later, on 24th December.

Apart from the usual illnesses of childhood, and anæmia when 18 years old, she had always been healthy. Menstruation began at the age of 15; was of the twenty-eight day type, and had always been regular. Duration five to seven days, and always rather profuse. She menstruated last on 11th October 1913.

She never suffered from dysmenorrhœa, but ever since puberty there had been a slight leucorrhœa for ten days after the menstrual flow stopped. For two months prior to admission there had been some irritability of the bladder, but not more marked than is frequently present in early pregnancy. The pain in the right side was more or less constant, and always worse on any exertion. Owing to the sickness and pain the patient had been confined to bed for about two weeks.

A few days after admission I made a careful examination under an anæsthetic, but was unable to arrive at a definite diagnosis, though I decided it was not a case of ordinary tubal gestation. I inclined more to the view that the uterus was fixed in the left side, and that it was expanding upwards and to the right side. What I found difficulty in explaining was the bulging downwards on the right side. This bulging in

the right fornix simulated a broad ligament cyst, and I was uncertain whether indeed I had not to deal with this condition complicating an ordinary pregnancy.

As there were no urgent symptoms the patient was kept under careful observation for a month, during which time she was examined on several occasions by Dr Barbour and myself. The swelling on the right side gradually extended upwards till it formed a well-marked elongated tumour reaching up into the lumbar region above the crest of the pelvis on the right side. Its walls seemed very thin, and on bimanual examination the uterus or what seemed to be the uterus lay below, and was pushed over to the left. No definite sulcus could be made out between the swelling and the fundus of the uterus. The diagnosis of pregnancy in an undeveloped right horn of the uterus was made, and owing to the tenderness and pain and possible rupture it was decided to operate.

On 3rd February 1914, when the pregnancy was advanced to about four and a half months, I opened the abdomen, Dr Barbour kindly assisting me at the operation. The condition found seemed to confirm the final diagnosis we had made of pregnancy in an undeveloped and isolated horn.

The right half of the uterus formed the swelling before described. Its wall was extremely thin and its lower part extended well out to the pelvic wall, opening up and expanding the broad ligament on that side. The left half of the uterus was solid, apparently well developed, and looked like a somewhat small uterus in its non-pregnant state.

It was impossible to determine whether we had to deal with a pregnancy in one half of a septate uterus or a pregnancy in an undeveloped isolated horn, due to a migration of the sperm from the opposite Fallopian tube; but the conditions found, the thinness of the gestation wall, the very marked irregularity of the growth of the right side of the uterus and its bulging downwards between the layers of the broad ligament,

all seemed to point to the latter, and it was decided to remove the whole uterus.

(In passing, I may say I had in mind a specimen I showed to the Society two or three years ago, which consisted of a maldeveloped uterus. The right horn was isolated and was the seat of a menstrual effusion of blood which caused the most severe dysmenorrhœa I have witnessed. This patient was a young girl of 14. I pass the specimen round again.)

The uterus was removed supra-vaginally, some difficulty being met with on the right side, owing to the opening up of the broad ligament there and the consequent small amount of space at the side of the pelvis.

On cutting through the cervix at the level of the internal os, it was found to have a double canal, each occupied by a separate plug of mucus.

Our diagnosis was, therefore, not strictly correct. We were dealing with, instead, a complete septate uterus, the septum extending right down to the internal os. Below this the development was normal, as there was a single external os and no trace of any vaginal septum. The specimen was hardened in formalin and then cut in a vertical transverse plane. It shows very well the points I have referred to, the complete septum, the pregnant right half with its very thin walls, especially so in its lower part where it opened up the broad ligament, and the non-pregnant left half of the uterus. The placenta, it will be noticed, is attached to the upper part of the uterine wall and not to the septum. Microscopic examination revealed a well-marked decidual reaction in the mucosa of the empty half of the uterus.

The patient made a perfect recovery and left the Hospital on 24th February, three weeks from the date of the operation.

Many cases of pregnancy in a septate uterus—uterus septus or sub-septus—are on record, and the condition is probably more common than we are aware of, as many have only been

discovered accidentally during labour or afterwards in multiparous women. I put on record in our *Transactions* a case of this sort in 1906, where I removed an abortion from the one half of the uterus and a fibroid polypus from the other.

In this case the patient had twice previously aborted, and this is a not uncommon history in such cases, though in the majority of the recorded cases no interference with the course of pregnancy has occurred. Abortion, it is stated, is generally the termination of those cases in which the placenta is unplanted on the septum. Labour as a rule is normal; cases, however, are on record where the hypertrophied non-pregnant horn has been displaced downwards and given rise to serious dystocia. Nagel records three such cases, and in Wohlein's and Bettman's cases the non-pregnant horn obstructing the pelvic cavity was the cause of the uterus rupturing during labour.

Difficulty and delay may arise also from lack of muscular force in the pregnant horn; the wall of the uterus, according to Winckel, becoming thinner and less capable of expansion in cases of arrested development of one or both Müllerian ducts.

Rupture of the uterus has also been recorded as occurring spontaneously during labour where no obstruction was present, and it can easily be produced spontaneously as in cases recorded by Winge and Faye and Robertson. Difficulty and delay may also arise from failure of the cervix, which is often rudimentary, to dilate, and from the oblique position of the pregnant horn preventing the expulsive forces being directed in the axis of the pelvis.

The very radical treatment adopted of removing the uterus in my case is, I am aware, open to criticism.

It must be remembered, however, that I was of the opinion at the time of the operation that I was dealing with a pregnancy in a hollow secondary isolated horn of the uterus, and in this view Dr Barbour concurred. Had the possibility of a septate uterus been thought of, the matter might have been determined

by passing a sound into the uterus at the time of the operation, but even then, in view of the very marked distortion of the uterus, the extremely thin wall, the expansion and special thinness of the lower part of the pregnant horn, not to mention the risks I have referred to in such cases, I feel sure the best was done for the patient by the complete removal of the uterus which was performed.

———

The President thanked Dr Fordyce for his paper, and asked whether there had been any menstruation during the pregnancy, to which Dr Fordyce replied in the negative. He also asked how one would be able to differentiate it from an interstitial pregnancy.

Dr Berry Hart said they were deeply indebted to Dr Fordyce for his paper. He did not think a nearer diagnosis could have been made, and he considered removal was quite justifiable.

Dr Ballantyne agreed that it was a very valuable paper, because it helped to lead to uniformity in the management of these cases, such uniformity being of slow growth because the cases were so rare. He referred to a case of uterus didelphys in which Cæsarian section had been done, and said that a case of angular pregnancy would be difficult to diagnose from one in a bicornuate uterus.

Dr Fordyce, in reply, said he ought to have mentioned that Dr Johnstone should have the credit for being on the right track in the diagnosis, for he had said it might be a pregnancy in an undeveloped horn.

III. A NEW ROUTE OF INQUIRY AS TO THE NATURE AND ESTABLISHMENT OF THE TYPICAL *SEX-ENSEMBLE* IN THE MAMMALIA.

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"*La Nature est intentionelle dans son but, mais aveugle dans l'execution.*"
Claude Bernard.

I.

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18 *Summary.*—Sex established early in phylogeny by the formation of gametes from protozoa, *i.e.*, primitive germ and sperm cells outside an organism; the human genital duct elements due to successive polar-body losses of the autonomous determinants causal to the unit characters, combined with their establishment pure and in default in the heredity cells; natural selection as an eliminant (pp. 314-19).

1. Preliminary.

A few words must be allotted to the question of the object of this inquiry. It is not, strictly speaking, an inquiry into the origin of sex in the mammalia, as sex had been established long before the appearance of this division of the animal kingdom, if we accept, as we must, the sex gland and its heredity cells as the final and only criterion of sex. What is here specially considered is the evolution of the *sex-ensemble*, i.e. of that portion of the body known as the urogenital tract when developing ontogenetically, or as the urinogenital tract when actually formed, with the secondary sexual characters.

The *sex-ensemble* thus comprises not only the sex glands, but the duct portion of the tract, the opposite sex-duct elements and the secondary bodily and psychical conditions associated with the *sex-ensemble*.

This distinction between *sex* and *sex-ensemble* must be kept in mind, otherwise confusion will arise. It must therefore be specially noted on this distinction that the question of sex, its nature and origin, will arise only incidentally, but that the

origin and nature of the *sex-ensemble* is the broad subject to be considered.

In the mammalia the organs concerned with reproduction have been most thoroughly investigated in the human male and female, but less accurately and fully in other mammals. In all mammalia it is the potent or characteristic organs that have been most fully examined. The genital organs contain, however, traces of an opposite sex-duct element, and thus the whole tract is like a clearly-written manuscript with a palimpsest portion, a partially erased section, hitherto little studied but of the greatest significance. It is thus to the potent urogenital tract, to the partially erased portion, to the secondary sex characters, and to the mammæ that I apply the term *sex-ensemble*. We must regard a mammal as made up of a soma and a sex gland containing the heredity cells and certain accessories. The soma is for the individual alone, while the future progeny are derived from the union of the reduced heredity cells. It is the union of these in the shape of gametes that gives rise to the fertilised ovum which is to express the heredity of the strain and of the race, and we must therefore, in the first place, consider how these heredity cells arise, how heredity is expressed in them and how variation, their great manifestation and result, is brought about.

As the zygote (fertilised ovum) is formed by the union of two gametes, female and male (ordinary ovum and spermatozoon), and as these are derived at maturation from the heredity cells when the polar bodies on each side are thrown off, we must now go on to consider—

2. The Origin of the Heredity Cells: The Male and Female Gametes.

By *heredity cells* we mean cells of that part of the zygote early set aside to form the primitive germ-cell mass. These travel through the developing embryo as early as the germ

layer stage, and ultimately reach the genital ridge on the Wolffian body and form with it the sex gland. When the heredity cells are thus *intruded* into the somatic part of the sex gland they form with it either an ovary or testis. In the ovary they are then termed oöcytes; in the testis, primitive sperm cells.

It is probable that in each sex there are two kinds of heredity cells, with the same number of chromosomes respectively. For reasons given afterwards they may be called in each sex Wolffian and non-Wolffian heredity cells. When the heredity cells at maturation have only half of their chromosomes prior to fertilisation, they are gametes.

It was van Beneden who first described the fact that the fertilising gametes had their chromosomes reduced to one-half by the loss of what were termed the polar bodies. This loss, known as "maturation" or "ejection of chromosomes," was a discovery of prime importance.

When the heredity cells reach the sex glands, mitosis begins, and its nature will be discussed presently. The discussion as to the origin of the heredity cells has been long and keenly carried on, but thanks to the work of Richard Owen, Eigenmann, Beard, Boveri, Lewis, Allen, King, Rubaschkin, and the advocacy of A. Keith, Felix, J. W. Jenkinson, the author, and others, the early zygotic origin is now supplanting the germ epithelium view. The view of the zygotic origin of the heredity cells is one of the greatest advances made in embryology, and clears up questions of heredity in a more simple manner than formerly.

The origin of the heredity cells from the germ epithelium of the ovary makes the former, with the determinants, arise from a somatic cell which has only determinants for its own class of cell. It makes the whole body of the adult somatic instead of the sum of the sex gland and the somatic part of the body.

This zygotic origin is further of prime importance, inasmuch as it is held by many that a somatic variation due to environment, etc., will be conveyed to the progeny. It can only so pass by first being made causal in the germ plasma, and there has as yet been described no mechanism by which a somatic variation can be made causal in the heredity cells.

3. Weismann's view as to Determinants of Zygote.

We may regard the part of the zygote set aside for the soma as containing the determinants for the body organs.

The heredity cells not only differ in their determinants, but are of two kinds. The one has Wolffian body determinants, the other none—the Wolffian and non-Wolffian heredity cells. The Wolffian relics differ in each sex, as the testis has more Wolffian than the ovary. When fertilisation takes place, it is either the union of a Wolffian male and a non-Wolffian female gamete giving a male zygote, or of a non-Wolffian male and a Wolffian female gamete giving a female zygote, and thus sex is determined at fertilisation.

In the nucleus of the heredity cells we have the chromosomes, and their electrons [1] are to be considered as being Weismann's determinants. There are no strict sex determinants in the chromosomes of the zygote. The heredity cells are, however, lodged ultimately in the genital ridge of the Wolffian bodies, and these latter differ in the two sexes in the portion they give to the formation of the sex tract. Thus in the male we have the whole Wolffian duct becoming the vas deferens, whereas in the female the Wolffian tubules and duct are only represented by the epoöphoron, the duct itself being rudimentary. Then, again, in the testis some of the Wolffian tubules become effective parts of its structure.

[1] See section 14.

Maturation.

In maturation, determinants are thrown off so that only one half remains. Those thrown off may be regarded as mainly ancestral, with a few immediate; those retained as mainly immediate, with a few ancestral. There are no direct sex determinants, *i.e.*, determinants for the oöcytes and sperm cells in the somatic part of the zygote, actual sex being due to the sperm cells and germ cells from an early division of the zygote, and these migrate as already described, until they become lodged in the genital ridge, and ultimately in the ovary or testis. Thus the heredity cells themselves, when in the sexual gland, determine the sex. If we have non-ciliated heredity cells in follicles, the sex is female; if motile and in tubuli, the sex is male.

With each sex there is, in mammalia, associated a typical duct system; in the female for the downward passage of the ovum, for the reception of the male seminal ejaculation, and also for the development and ultimate expulsion of the developed fœtus; while in the male there is an excretory duct for the passage of spermatozoa, and an intromittent organ with accessory glands for sexual congress. Whether the vesiculæ seminales store the sperm cells is not as yet accurately settled.

In each sex there thus is a potent or normal genital tract, and a non-potent or opposite sex-duct element. The latter are, *in the female*, the epoöphoron and paroöphoron; *in the male*, the hydatid testis and prostatic utricle (see Plate I., Figs. 1 and 2).

4. The Urogenital Organs exercise the function of Sex, as the Heart and Blood-vessels, for instance, carry on that of the Circulation of the Blood.

A special system of the body, the urogenital, is set aside in mammalia for the function of sex, just as we have special systems

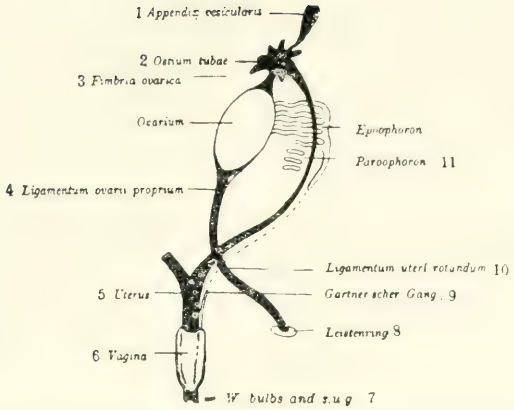

FIG. 1.—Typical female *sex-ensemble* (modified from Bonnet), shows (1) *Sex-gland* (ovary); (2) *Potent portion*—Tubes, uterus, vagina and external genitals; (3) *Non-potent or opposite sex-duct elements*—Epoöphoron and paroöphoron 11; secondary sex characteristics not indicated (see pp. 281 and 282).

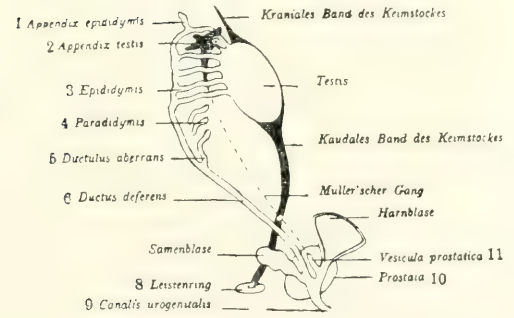

FIG. 2.—Typical male *sex-ensemble* (from Bonnet), shows (1) *Sex-gland* (testis); (2) *Potent portion*—Vas deferens and epididymis, gubernaculum; (3) *Non-potent or opposite sex-duct elements*—Hydatid testis, prostatic utricle 11; secondary sex characteristics not shown (see pp. 281 and 282).

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formed in the soma for the cardiac, respiratory, digestive, and intellectual processes. The urogenital organs, characteristic for each sex, form for reproduction that system in mammalia, and it is the phylogenesis of this system that is the key to the question of sex and of the sex-ensemble.

We must speak, however, of the characteristic *sex-ensemble* instead of the characteristic "sex."[1] By the term "*sex-ensemble*" one understands not only the actual sex gland and its potent ducts, but also the opposite sex-duct elements; the mammæ, type of pelvis and body, arrangement and distribution of hair, and special psychical qualities. There is no more reason for believing that the typical *sex-ensemble* in mammalia is suddenly developed at fertilisation, than that the same happens to a four-chambered heart or to a human brain. Each has its long-drawn-out phylogeny, and its ontogeny is the culmination of this in the special organism whose development is being considered.

We have now to take up, mainly from a morphological aspect, the nature of the genital tract in the human female and male.

5. What makes up in detail the Typical Sex-ensemble.

It comprises the following structures in each sex:—

The Typical Female Sex-ensemble.

Potent.—Ovary, uterus, tubes, vagina, hymen, external genitals.

Non-potent or Opposite Sex-Duct Elements.—Epoöphoron and paroöphoron.

[1] One uses urogenital as embracing the Wolffian bodies, their ducts, and urinogenital sinus, while the urinogenital organs are the fully-formed organs, and arise from the former (see as to exact origins, p. 277).

Secondary Sexual Characteristics.—Mammæ, characteristic pelvis, pubic hair distribution, larynx, special psycho-sexual development (Plate I., Fig. 1).

The Typical Male Sex-ensemble.

Potent.—Testes, vas deferens, phallus.

Non-potent or Opposite Sex-Duct Elements.—Hydatid testis, prostatic utricle (Plate I., Fig. 2).

Secondary Sexual Characteristics.— Characteristic pelvis, distribution of pubic hair, mammæ, larynx, psycho-sexual attributes.

In each typical sex-ensemble the potent or characteristic organs are at their maximum, the opposite sex-duct elements at their minimum; the secondary sexual characters congruent. Any ratio disturbance of this makes an atypical sex-ensemble.

6. The Development and Origin of the Female Urinogenital Tract, and of the Opposite Sex-Duct Elements in it.

The foundation of the urinogenital tract is the excretory one known as the Wolffian system, made up of the pronephros with its duct, the pronephric duct—both practically gone—and the mesonephros and its duct, usually termed the Wolffian duct —both fully represented in the early embryo.

The Pronephros with its Duct.—It would be out of place to trace the phylogeny of this system. In the human embryo it is occasionally seen as a depression at the cephalic end of the Wolffian body with a short duct. Its duct may be the ancestor of the Müllerian duct, but this is uncertain.

The *mesonephros or Wolffian body proper and its duct* are present in the early human embryo, and act as temporary kidneys, although this is disputed, until the true kidney and its ureter (metanephros and duct) are developed, mainly as an outgrowth from the Wolffian duct.

On the Wolffian body there early develops a special longitudinal thickening continuous with the cœlomic epithelium, the genital ridge, and the latter with certain parts of the Wolffian body make up the somatic portion of the male and female sex gland.

The Wolffian duct opens in the embryo into the urinogenital sinus below the eminence of Müller, the blind end of the Müllerian ducts, as can be well demonstrated in serial sections of a six-weeks' female embryo.

It must, therefore, be strongly emphasised that the pronephric and Wolffian bodies and their ducts are the foundation of the genital system in mammals, and that their non-development or loss will profoundly modify it.

In the genital ridge of the Wolffian bodies the ovary and testis arise, and contain the well-known oöcytes and sperm cells —the former in follicles, the latter in tubules—as has been already considered (pp. 266, 267). In the case of the oöcytes, Graafian follicles form round them. Before this stage they are primitive oöcytes with an envelope of spindle cells surrounding each of them.

The nucleus of the heredity cell contains the chromosomes in the shape of microscopic rods, which stain readily with aniline dyes, and have Weismann's determinants of heredity in them. It is the chromosomes that are the active part of the nucleus, as we shall see when we take up the questions of mitosis and maturation. The rôle of the cytoplasm is not settled.

The *Fallopian tubes, uterus, and vagina* arise from the Müllerian ducts. These are two in number, one on each Wolffian body, but become differentiated afterwards by disappearance or persistence of their septal walls into the Fallopian tubes and a single uterus and vagina (in the human female). Their phylogeny is obscure, but if they are not pronephric in origin each is most probably a modification of such a primitive

duct system, as the pronephros in lower organisms communicates with the cœlom above and the urinogenital sinus below.

The actual Müllerian ducts in the human female arise as a thickening of the cœlomic epithelium on the mesial or lateral aspects of the Wolffian bodies in 7·5 mm. embryos.

In the human embryo the Müllerian ducts open at first blindly at the Müllerian eminence on the posterior wall of the urinogenital sinus above the patent orifices of the Wolffian ducts proper.

The lowest third of the vagina is formed from the urinogenital sinus along with the lower ends of the Wolffian ducts, and the latter develop the hymen. The upper two-thirds are Müllerian.

The question of the origin of the vagina and hymen may, however, be discussed more fully.

The usual view is that the vagina and hymen are derived entirely from the ducts of Müller, while all that represents the urinogenital sinus, apart from what gives rise to the urethra, is the vestibule. In the six weeks' fœtus, however, the urinogenital sinus is very long, and one has also to account for the multilayered lining of the vagina. The early embryo after the fourth week has the ducts of Müller coalesced, and the lining of the future vagina is of a low columnar type.

Quite a new aspect was given to this question when it was found that about the third month the vaginal lumen was solid (Klein, Nagel), and that at the future site of the hymen there was a bulbous proliferation of epithelial cells of the type found in the adult vagina. In investigating a female fœtus at the third and a half month I found two distinct and lateral bulbs composed of a similar epithelium, and traced the Wolffian duct into one of them. The Wolffian duct is of ectodermal origin (Kollmann), and thus the hymen is really an organ of the urinogenital sinus and of Wolffian duct origin. Strange to say, I found quite a similar bulb in the pros-

tate of a four-months' male embryo in a line with the vas deferens.

The nature of the vaginal and vestibular tract I believe to be as follows: — The upper two-thirds of the vagina are Müllerian, the lower third urinogenital sinus, while the vestibule is also derived from the urinogenital sinus.

As corroborative of this it may be noted that the lymphatics of the lower third of the vagina pass to the groin glands, those of the upper two-thirds go with the uterine ones. The muscular, nervous, and blood supply of the lower vaginal third also differ from that of the upper two-thirds.

In the three and a half months' vagina there is no lumen, but the lumen is filled up with cells bordered by a narrow clear line. These blocking cells are continuous with those in the bulbs, and evidently these bulb-cells have passed into the Müllerian canal and absorbed the primitive low columnar epithelium. This irruption passes up as far as the lower part of the cervical canal, and ultimately desquamates centrally, so that the ultimate vaginal lumen is thus formed. Those who hold to the entire Müllerian origin suppose that the primitive epithelium becomes changed into a squamous form.

Origin of the Bladder and Urinogenital Sinus.—By a ventral unfolding of the caudal blastoderm in the 2 mm. embryo we get the lower part of the primitive gut, the entodermal cloaca and tail gut, formed. The former, the pars penultima, becomes, by a coronal partition dipping from above, divided into two parts, the anterior forming the bladder, the posterior the future rectum. The allantois takes no part in forming the bladder, although it is traditionally held in respect by many anatomists and used as the *deus ex machinâ* in extroversio vesicæ, but only the fundus vesicæ may be considered as allantoic. There is no free or vesicular allantois in the human embryo, and it is only represented in the umbilical cord by an imperfect duct near the navel.

The *tail gut* (pars ultima) disappears early, but I have recorded a unique case where it was present attached to the anterior aspect of the perineum of a healthy female child. It was in appearance like a scrotum, had an aperture on its anterior aspect, and was lined with Lieberkühnian follicles. This shows that the tail gut has also a cloacal membrane. The tail gut may be called the pars ultima of the primitive gut.

The *urethra* in its upper part is derived from the urino-genital sinus. The lower part is blocked, like the vagina, by a proliferation of epithelium from the Wolffian bulbs, and is retunnelled like the primitive hymeneal site. The urinogenital sinus thus forms the urethra, the lower third of the vagina and the parts inside the labia minora, viz., the vestibule and the part of the fossa navicularis between the fourchette and hymen, the former being due to the junction of the lower ends of the labia minora.

The Cloacal Membrane.—When the pars penultima of the primitive gut is formed, its anterior mesial line, in direct descent, probably from the gastrula pore and neurenteric canal, closes in at its ectodermal aspect, and then mesoderm grows in to complete the bony and muscular ring. The ectoderm alone may be present or all the layers may be absent, a defect which, when complete in its linear extent, gives rise to extroversio vesicæ. Minor amount of non-union gives epispadias.

The *hymen* is not the thinned-out ends of the Müllerian ducts, but is due to a bulbous epithelial proliferation of the lower ends of the Wolffian ducts; the coalescence and central breaking up of the bulbs aided by an involution from below forms the vertical slit-like entrance of the hymeneal aperture.

There are other normal structures in the female genital tract which must now be considered.

The *broad ligaments* represent the greater part of the Wolffian bodies.

The *ovarian ligament* is the equivalent of the caudal ligament of the testis seen in the early male embryo while the testis is still undescended.

The *round ligament* is the equivalent of the gubernaculum testis at the stage when the testis is still abdominal.

The *rudimentary structures* between the layers of the broad ligament—the epoöphoron and its duct and the paroöphoron—will be considered presently.

In a certain proportion of cases *suprarenal tissue* is found in the broad ligaments and near the testis.

It is interesting to note that in chrysemys embryos a number of heredity cells pass dorsad above the root of the mesentery until they come to lie in the loose mesenchyme immediately beneath the aorta and often slightly to either side of the anlagen of the adrenal bodies (Lewis). This means, then, that in the so-called descent of the testes in which the heredity cells lodge, the suprarenal may in part also descend. This descent is not constant but occasional, in this differing from the constant co-existence of the epoöphoron with the ovary. It has also been found that the upper limit of the migration of the heredity cells is at the pronephros, and its descent as the hydatid testes is thus better understood. It indirectly confirms the origin of the fimbriated end of the Fallopian tube from the pronephric funnel, as F. M. Balfour and A. Keith have stated.

The Opposite Sex-Duct Elements in the Typical Human Female Genital Tract.

In the human female genital tract there are present not only the organs characteristic of the *sex-ensemble*, but also those that are the degenerated analogues of certain parts of the male genital tract where they are fully developed. The former have been termed the potent organs, the latter the non-potent, or,

more exactly, the opposite sex-duct element. The latter are degenerated segmental tracts or parts, analogues to those normally developed in the male genital potent tract, and are known as the epoöphoron (parovarium) and its duct, and paroöphoron-tubules lower down at the hilum ovarii.

Since these opposite sex-duct elements in the genital tract were first pointed out by Rosenmüller in 1802 and Kobelt in 1847, little has been done in the way of their investigation, but there have been notable additions to our knowledge by Gartner of Copenhagen (1824), Numan of Utrecht (1843), Meyer of Berlin (1897-1913), Arthur Keith, and Alban Doran. Rosenmüller's account is excellent, and his figures of the epoöphoron accurate.

Kobelt gives a valuable description of the opposite sex-duct elements, and an interesting account of atypical sex in a goat (a male really), not one of twins apparently, and where the testes had descended. His drawings, which are very fine, have been repeatedly copied in text-books.

Gartner's description of what is known as Gartner's canal I have already given from Numan's work. Meyer of Berlin has written comprehensively (1897-1913) on the subject of the representation of Gartner's canal—the Wolffian duct—in the human fœtus. Plate II., Fig. 1, shows its relation to the vagina and uterus schematically, while Fig. 2 gives a case where it ended in the hymen, an interesting fact in relation to the view I have advanced as to the origin of the hymen from the Wolffian ducts and not from the Müllerian, as is generally held.

Numan found great difficulty in tracing Gartner's ducts in sheep, as they are, so far as is yet known, little developed. He used mercurial injections to demonstrate them in the cow. It is this opposite sex-duct element that is the almost completely obliterated palimpsest of the genital tract—with a complete understanding of its nature is linked the clearing up of the

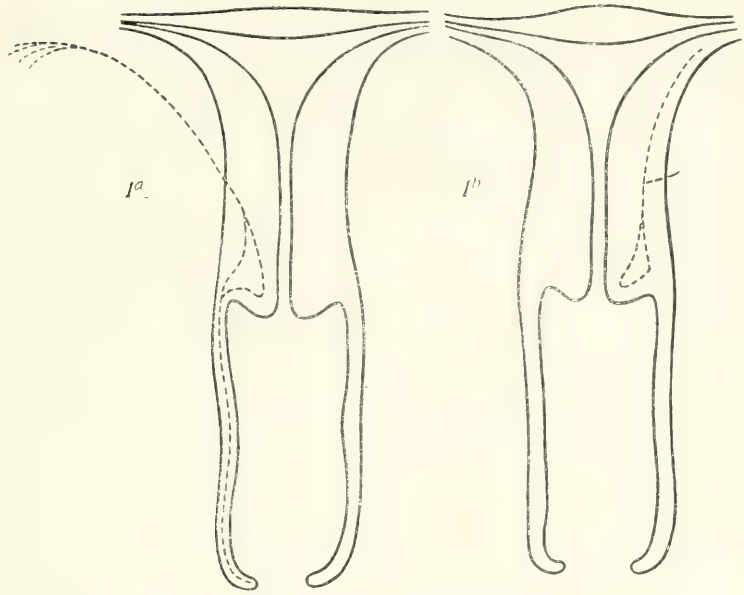


FIG. 1.—Schematic frontal section through uterus and vagina.

The Gartner canal is shown as a dotted line in the broad ligament, parametrium about the level of the os internum; the ampullary dilatation in the cervix is seen as well as the passage of the canal in the vaginal wall down to the hymen. (Meyer.)

FIG. 2.—Longitudinal section through the opening of Gartner's canal in the hymen.

Gartner's canal, *G.*; hymeneal epithelium, *H.E.*; *H.B.*, connective tissue. From the vulva of a new-born infant. (Meyer.) Note difference of epithelium in the part of the duct in the hymen.

[Face page 276.

nature of the normal *sex-ensemble*. We may now give the developmental origin of the urinogenital system.

Summary of Source of Female Genital Organs.

Organs.	Source.
Heredity cells.	Early division of zygote.
Somatic part of ovary.	Genital ridge and connective tissue of Wolffian body.
Fallopian tubes, uterus, and vagina (upper two-thirds).	Müllerian ducts.
Lower third of vagina.	Upper part of urinogenital sinus and lower ends of Wolffian ducts.
Hymen.	An organ of the urinogenital sinus and derived from the Wolffian ducts.
Vestibule.	Lower part of urinogenital sinus.
External genitals.	External region on and below pubes (imperfectly worked out).
Bladder.	Anterior division of entodermal cloaca ; fundus allantoic.
Urethra.	Upper part from the urinogenital sinus, also lower part, but it is retunnelled.
Ureter.	Upward growth from Wolffian duct.
Kidney.	Upward growth, in great part from Wolffian duct, an upper part from Wolffian body.

The next step is to show that the genital tract is not to be looked on as a continuous one unsegmented in its origin, but segmental, and the proof of this is sought in an analysis of deformities that are exact. To use a simile, it is not like a concrete building, continuous and unsegmented, but one built up of separate portions—like a brick edifice. We analyse now, therefore, the exact deformities, the lost bricks, and base on this an understanding of its normal elements or unit characters.

7. The Exact Deformities of the Female Urogenital Tract in relation to its Time Development.

We may have exact persistent segmental fœtal forms in adult life:—

(1) Complete defect of both Müllerian cords, and therefore absence of uterus, tubes, and vagina. (2) Complete absence of anlage of a single Müllerian cord (uterus unicornis verus). (3) Complete separation of both Müllerian cords (uterus didelphys or duplex separatus). (4) Absence of cavity formation in separated and united Müllerian cords, uterus rudimentarius solidus, duplex, bicornis, simplex cum vagina solida. (5) Partial cavity formation in the separate or united Müllerian cords, uterus rudimentarius partim excavatus, duplex, bicornis, simplex cum vagina solida. (6) Uterus bicornis with rudimentary development of a horn. (7) Uterus bicornis septus, subseptus, simplex, vagina septa, subsepta simplex. (8) Uterus introrsum arcuatus septus, subseptus simplex, vagina septa, subsepta, simplex. (9) Uterus planifundalis septus, subseptus simplex, vagina septa, subsepta, simplex. (10) Uterus foras arcuatus, septus, subseptus, simplex, vagina septa, subsepta, simplex. (11) Uterus fœtalis. (12) Uterus infantilis et virgineus. (13) Uterus inequalis vel obliquus. (14) Hypoplasia uteri, as in chlorosis and anæmia (von Winckel).

These may be permanent in the adult. To these may be added—

(15) Atresia vaginæ; persistence of imperforate hymen in its usual site, or as a transverse septum an inch from the Wolffian hymen. The latter is the persistent eminence of Müller. (16) Persistence of solid cervix in lower third, causing hæmatometra and hæmatosalpinx.

It must also be noted that exact *segments* of the genital tract may be awanting as follows:—

Fallopian Tubes.—Total defect is seen most usually when the genital organs are not represented at all. Single absence

is found in the unicornis uterus; the lateral third has been noted as absent where the rest of the tract was normally developed. The middle third may be represented by a connective tissue rudiment. In a case recorded by W. G. Spencer and Alban Doran the tubes alone were unrepresented, while the uterus, ovaries, vagina with hymen, were present. The fimbriated end of the tube was represented by traces in the ovarian pouch (ovarian fossa), and this Doran explains on F. M. Balfour and Keith's view that this part of the tube is related phylogenetically to the pronephros. The round ligaments were present.

We have next to consider cases where, with local losses in the urogenital tract, there are associated loss conditions of a more extensive nature.

In a recent paper by Bolaffio, where he records a case of uterus bicornis with septate vagina, one-half being rudimentary, with defective urethra and defect of the right half of the trigone of the bladder, he classifies 99 cases of *conjoined* defects in the urogenital system as follows :—

Tabular result of 99 Cases of Genital and Renal Anomalies.

Uterus unicornis sine rud. cornu alt	13	Unilateral complete absence of kidney	12
		Rudimentary kidneys	1
		Congenital ectopy of kidneys	0
Uterus duplex separatus cum vagina duplice separata	1	Unilateral complete absence of kidney	0
		Rudimentary kidneys	0
		Congenital ectopy of kidneys	1
Uterus rud. solidus oder partim excavatus	19	Unilateral complete absence of kidney	10 (in 7, also ectopy)
		Rudimentary kidneys	0
		Congenital ectopy of kidneys	16 (in 7, also absence of kidney)
Uterus unicornis cum rud. cornu alt	24	Unilateral complete absence of kidney	19 (twice ectopy of kidney)
		Rudimentary kidneys	1 (ectopic)
		Congenital ectopy of kidneys	8 (twice, also absence and once rudimentary)

TABULAR RESULT OF 99 CASES—*continued.*

Tube defect alone	3	Unilateral complete absence of kidney	3
		Rudimentary kidneys	0
		Congenital kidney dystopy	0
		Lateral complete absence of kidney	25
Uterus bicornis septus and subseptus	32	Rudimentary kidneys	6 (7 times ectopic)
		Congenital kidney dystopy	3 (twice bladder kidneys)
Uterus introrsum arcuatus and planifundalis septus, subseptus simplex	6	Unilateral complete kidney absence	3
		Rudimentary kidneys	2
		Congenital kidney dystopy	3 (thrice rudimentary)

In addition to the above disturbances of the sex tube and penultimate gut, we may have the *ovaries* abnormal. Both may be awanting in general absence of the genital organs. It is very rare, if not impossible, to have one ovary alone awanting and the rest of the tract present.

8. THE MALE UROGENITAL SYSTEM: ITS INDIVIDUAL COMPONENTS THAT MAY BE LOST; LOSS OF THE WHOLE SYSTEM IN WHOLE OR IN PART.

Preliminaries as to Development.

In the human male the bladder, rectum, and kidney develop as in the female; so also, probably, the heredity cells (sperm cells). The external genitals are special developments from the pubic and subpubic ectoderm and mesoderm and from the cloaca. The scrotum is the equivalent of the labia majora, and the closed urethra is represented by the labia minora and urethra of the female. The testes are normally in the scrotum, *i.e.*, have advanced to a complete descent from one represented by the permanent pelvic position of the female. The condition of the neck of the bladder is markedly different from that of the female. In the former the prostatic urethra is a comparatively narrow tube surrounded by the well-developed prostate, represented in the female only by Skene's tubules.

In the prostate we have the prostatic utricle, where the vasa deferentia open (mesonephric ducts), the former representing the hymen. Attached to the testes is the hydatid testis, the rudimentary representative of part of the opposite sex-duct element, the fimbriated end of the Fallopian tube. Thus the mesonephric duct is fully represented in the male, while the structures forming the Müllerian ducts of the female are represented only by rudimentary elements (hydatid testis).

The *sex gland* (testis) develops its framework on the genital ridge of the Wolffian body, utilises parts of the ducts for its tubular portion, taking the Wolffian duct proper for its *vas deferens*. The sperm cells probably originate from an early division of the male zygote and travel to the genital ridge of the Wolffian body, but all this has not yet had so full and demonstrative investigation as it has obtained in the analogous process in the female (see p. 277). It is quite possible that in the early progress of the heredity cells along the germinal path in the germ layers we are unable to distinguish male from female heredity cells.

The Individual Gross Components of the Male Urogenital Tract that may be Lost; Loss of the Urogenital System in whole or in part.

The exact defects of the male urogenital tract have not been worked out so fully as in the case of the female. This may be due to the greater simplicity of the tract and to a relative infrequency of malformations.

The male urogenital system may be completely lacking on one side, and this has been described by Professor van den Broek of Utrecht in an interesting paper, "Ein Fall von vollkommener Agenesie des rechten Urogenitalapparates," giving also a series of cases where there were full or partial defects. In his special case the aorta, vena cava inferior, and the viscera were normal. There was no special right renal

artery or vein, and the right kidney was entirely absent without any rudiment even in the connective tissue where the kidney normally lies. The ureter was awanting.

The left kidney was abnormally large (15 cm. × 8 cm. × 4 cm.). The pelvis was single as well as the ureter, and the latter opened normally into the bladder. There was no trigone, but a marked ridge ran between the ureteric opening and the colliculus seminalis. There was no vas deferens or vesicula seminalis on the right side, and the right side of the prostate was so small that the whole gland was asymmetrical in shape. The suprarenal was not in its position, but by tracing the sympathetic it was found in the pelvis to the mesial side of the psoas muscle and somewhat beneath it.

As to the external genitals, the scrotum was small and contained only one testis, in the middle line behind the phallus. There was also a pigmented mesial scrotal streak. Other cases are given, and the results tabulated as follows:—

1. In lateral absence of the kidney we may have total want of the corresponding urogenital tract, apart from the phallus.

2. Testis present; epididymis, vas deferens, vesicula seminalis, ureter, and kidney absent.

3. Testis present; epididymis partially present or defective; vas deferens, vesicula seminalis, ureter, and kidney awanting.

4. Testis, epididymis, and vas deferens present; vesicula seminalis, ureter, and kidney awanting.

5. Ureter and kidney alone awanting.

6. Ureter ending blindly above; kidney awanting or rudimentary.

7. Congenital cystic kidney. Here there is probably a want of union between the part of the kidney developing from the Wolffian duct and that laid down higher up.

In regard to the descent of the testes, we may have the gubernaculum awanting, and a small non-descended testis as in the free-martin in cattle; or the presence in the human male

of the testes in the abdomen and pelvis, non-descent of the testis. I have already discussed this question in another paper.

The male urogenital system is thus segmental—viz., made up of kidney, ureter, epididymis, vas deferens, vesiculæ seminales, gubernaculum testis on each side, and these segments may be awanting, on one side usually, either individually or as a whole.

The non-potent or opposite sex-duct elements are also segmented, and thus in man all these segments are lost and degenerated, except the hydatid testis and the prostatic utricle.

We see, therefore, that the male and female genital tracts, potent and non-potent, are made up of exact segments, and that these segments are also grouped in larger units. Both may be regarded as unit characters or grouped unit characters in a Mendelian sense.

Contrast between Male and Female Sex Organs.

The human male and female urogenital tracts are so constructed, or have so evolved, that each contains in its special sex and sex-duct portion, potent and non-potent organs, the latter in each case being segmentally defective and degenerated relics of the genital ducts of the opposite sex.

The most pronounced differences between the sexes are: (1) The pelvic position of the ovaries, the testes being extra-abdominal and in the scrotum. (2) The fully-developed and permanent round ligaments and ovarian ligaments represented in the male fœtus in the earlier months by the transient stages of the gubernaculum and testicular caudal ligament. (3) A split condition of the labia majora and minora comparable with the early stages in the male leading up to the formation of the scrotum, the closure of the spongiosum, and the formation of the urethra. We have, however, exact losses in the female

as compared with the male. (4) A loss of the middle segments of the Wolffian duct and of the greater part of the Wolffian body, but retention of a part of it as epoöphoron and paroöphoron as well as of the lower ends of the Wolffian ducts to form the hymen. The corpus spongiosum is almost entirely awanting, the cc. cavernosa partly in the female, and mainly in an aplasic state.

It is evident that the urogenital system is based on the Wolffian bodies and ducts. In the male the mesonephric duct is the main one; in the female a similar excretory duct—the potent tract—probably the pronephric. It is from or in connection with the Wolffian bodies and ducts and from the primitive gut that the urogenital organs arise, and thus any growth-disturbance or aplasia of the former leads to serious changes in the latter.

The human male and female may be looked on as "varieties" of homo, the variation being in the sex organs for the purpose of sexual reproduction. With this is linked a condition of internal secretory glands which lead to secondary changes in the mental attitude to the world and in actual bodily condition—in pelvis, subcutaneous tissues, and in the epidermis and its derivatives—skin, mammæ, mesial incisor teeth. The mental attitude in the female is passivity or a forced activity; in the male natural activity. The one seeks, the other is sought. The female intellect is more intuitive and subtle; the male more balanced and restrained. In the female the emotions lead; in the male they are much more under control.

The argument thus has been that the sex-duct organs are segmented, each with degenerated representatives of the opposite sex. We must now, therefore, trace more deeply how these sexual differences have arisen, and begin by discussing—

9. The significance of Mendel's work.

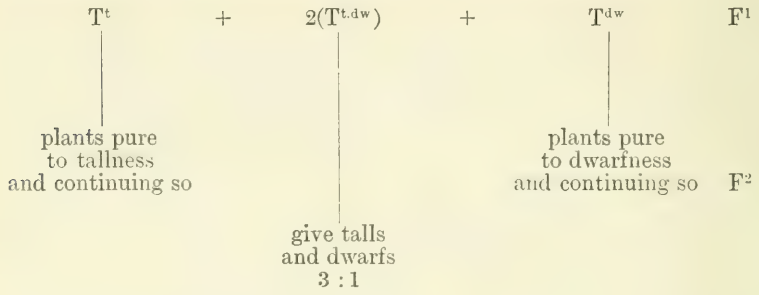
Mendel showed by his famous plant crossings that (1) the "soma" of a plant or animal is to be regarded as made up of autonomous parts—that is, of structures which interchange in themselves during succeeding generations but do not blend. These are termed unit characters; (2) when crossings are made in plants or animals with one or more contrasted and thus recognisable unit characters—tallness and dwarfness in peas, for instance—on each side, the ultimate result is that these contrasted qualities are noted to sift out in a probability ratio of 1 : 2 : 1 when there is one contrasted character on each side, of 9 : 3 : 3 :1 when there are two on each side, and so on; (3) in a crossing with one contrasted unit character on each, one of these is alone represented in the soma of the plants of the first generation (F^1), but afterwards both qualities come out in a probability ratio in the plants (*i.e.* in the soma), with no preference for one quality, *quâ* the soma, as in F^1. On this preference for one quality in the soma of F^1 a theory of dominance and recession or strength and weakness was formulated by Mendel and has been pushed by many observers, especially among English Mendelians. It would take too long to discuss the whole question, but the correct reading of a Tall × Dwarf crossing is as follows:—

If T and D represent the tall and dwarf plant soma and t and dw the causes or determinants of the tallness and dwarfness as expressed in the determinants of the pollen grains and egg cells, the scheme is as follows:—

$$\begin{array}{ll} T^t \times D^{dw} & P \\ \| & \\ T & F^1 \end{array}$$

i.e. plants all somatically tall.

In the pollen grain and egg cells, however, the selected qualities are distributed in a probability ratio as follows:—



The talls from T^t continue to breed true to tallness, while $2(T^{t.dw})$ gives in F^2 $\frac{1}{3}$ of pure talls, the others, so-called impure talls, again giving T to D 3 : 1. From T^{dw} pure dwarfs continue to come in subsequent generations. When the generations $F^1 - F^n$ are summed up as to plant results, we get a ratio of pure talls to impure talls 1 : 2 : 1, a probability ratio. There is only purity of gametes to a contrasted quality in those breeding pure to a quality. In the impure talls two-thirds have both qualities in the gametes.

The 1 : 2 : 1 ratio is not due, therefore, to a *probability combination* of pollen grains and egg cells, or gametes as they have been termed, but to a *probability distribution* of their contained determinants in the oöspore, *i.e.*, in the part of the oöspore from which the pollen grains and egg cells arise, the other part having only the somatic determinants in it.

Dominance and recession mean merely that in F^1 one contrasted quality appears in the *soma*, but both qualities are distributed causally and in a 1 : 2 : 1 ratio in the pollen grain and egg cells, and these when interfertilised give the 1 : 2 : 1 ratio in the somata of F^2. The result of the 1 : 2 : 1 ratio is that each of the qualities is placed pure for $\frac{1}{4}$ of the plants,

while $\frac{1}{2}$ give the 3 : 1 ratio[1] as already explained, and *thus a pure strain for each contrasted character established.* In one half the contrasted qualities are placed together in the pollen grains and egg cells or in the gametes, and the ratios given out as 3 : 1. This partial and fractional purity result is of the highest importance.

It is not enough to consider Mendel's unit characters as they are in the adult soma; we must go on to connect Mendel's results with Weismann's views, and I therefore now take up the question of—

10. How are we to regard Weismann's Determinants?

We may look on Weismann's determinants in the heredity cells as electrons of protoplasm, as hormones, with a part for the actual basis of the organs to which they are causally related, and we may speak of them as the causal determinants, or in brief as the determinants. On the view about to be laid down they may be considered as neutral molecules, and when under some stimulus causing loss and gain of electrons, as positive and negative ions, each becoming one or the other as the stimulus acts or ceases to act. The positive and negative ions are to be considered as acting under the convention that like ions repel, and unlike attract. It is in the molecular whirl of the determinants that atoms of protoplasm (electrons), with or without their basic substance, are thrown off—maturation at fertilisation—and we thus have the three possibilities of charged determinants, neutral molecules, or negative and positive ions. The most exaggerated whirl and extrusion of electrons, etc., is seen in radium, but we must regard living protoplasm as having this instability in a marked degree.

[1] The 3 : 1 ratio is due to the fact that the pure talls and those that give rise to some dwarfs and talls are the same somatically, and thus $\frac{1:2:1}{3:1}$ is the combination result.

One argues here from the actual organs in the body of man back to their causal agencies in the germ plasma, just as Dalton, in his celebrated atomic theory, reasoned from exact chemical interchanges of substances back to the atoms.

I now go on to apply the theory that the human genital tract is segmental in its nature, and that these segments are Mendel's unit characters; that they are causally represented in the heredity cells and their derivatives by Weismann's determinants. In the allotment of these determinants at fertilisation, I regard the determinants as distributed in the heredity cells of the p. g. c. mass in a probability ratio, while at maturation prior to fertilisation there is a loss of determinants in the ejection of the polar bodies and that in this way each one, or group, of any of the determinants may serially be cast off, causing discontinuous variation. In mitosis we have, as we shall see (section 14 *et seq.*), electrolysis and distribution of ions of protoplasm to the positive and negative parts of the cell (anions and kations), bringing about continuous variation. Finally, it is from polar losses that we ultimately get a probability ratio between the potent and non-potent tracts. At present embryologists are neglecting the molecular physics of mitotic and maturation changes prior to and at fertilisation, but must range themselves up with chemists and physicists in applying modern views to this stage of development. The molecular physics are specially considered under section 14, but we must first take up—

11. Recognition of the Unit Characters of the Genital Tract from previous analysis of Exact Deformities of Human Urogenital Tract.

From the exact losses in the male and female urogenital tract known as deformities, and from those producing the opposite sex-duct element in each sex, we may regard both

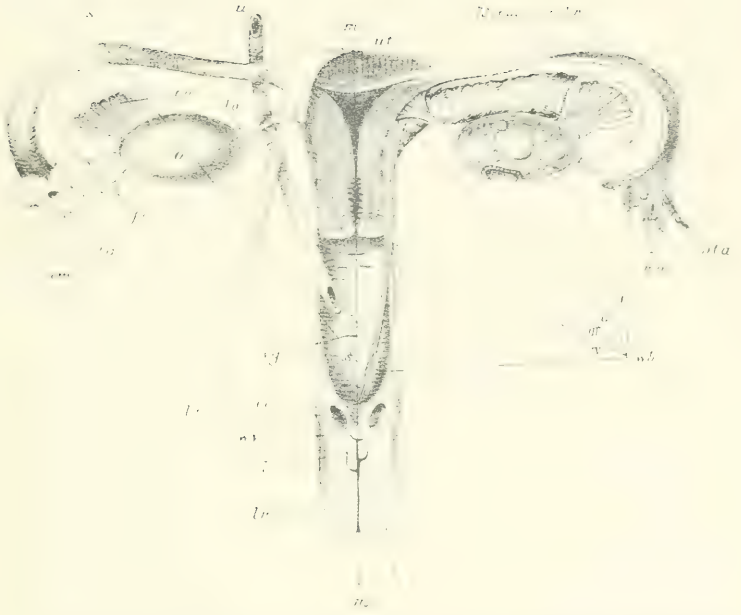


Fig. 1.—Female typical *sex-ensemble* shown on Coblenz's diagram ($\frac{1}{3}$). *Potent organs*—Ovary (*o*), tubes (*fo*, *ota*, *etc.*), uterus (*ut*), vagina (*vg*), external genitals (*cc*, *ve*, *l*). *Non-potent or opposite sex-duct elements* (*wb* I. to IV.)—Epoöphoron (I. and *po*), paroöphoron (II. and III.), Gartner's canal (II. to IV.), round ligament (*lr*), ureter (*u*). The secondary sexual characters not shown, but see pp. 269 and 270. The potent duct elements are at a maximum of segments, the non-potent at a minimum, and the secondary characteristics congruent to the sex.

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tracts as made up of certain segmental elements or unit characters of Mendel.

Unit Characters of the Potent Female Urinogenital Tract.—These require in the present state of our knowledge to be taken up in relation to the fully-formed organs. They might be selected, but not so easily, from the primitive basal organs, Wolffian bodies and ducts, Müllerian ducts, urogenital sinus, cloacal membrane, and primitive gut; but as the former are better investigated, and are likely to continue to be so in the future, they will be considered here, the basal structures, however, always being kept in mind.

These *unit characters* may therefore be considered as the following:—

Potent—

	No.
Ovaries	2
Mammæ	2
Fallopian tubes	6
Uterus (body and cervix)	4
Vagina (upper two-thirds)	2
Vagina (from urinogenital sinus)	1
Hymen	2
Vestibule	1
Larynx	1
Suprarenals	4
Round ligaments	2
Ovarian ligaments	2
Broad ligaments	2
Glans clitoridis	2
Prepuce	1
C. cavernosa	2
Absorbing factors as to septa in uterus	1
„ „ cervix	1
„ „ vagina	1
„ „ hymen	1
	40

Non-Potent or Opposite Sex Elements.—Epoöphoron and duct, 4. The ratio is thus 40 : 4.

THE SEGMENTED MALE UROGENITAL TRACT.

Potent—

	No.
Testes	2
Epididymis	2
Vasa deferentia	6
Vesiculæ seminales	4
Prepuce	2
C. spongiosum	1
C. cavernosa	2
Gubernacula	2
Suprarenals	4
Larynx	1
Absorbing factors in scrotum	1
	27

Non-Potent—

Hydatid testis	2
Prostatic utricle	2
Mammæ	2
	6

We thus get the ratio as 27 to 6 approximately.

The female urogenital tract may be thus considered as having a 40 : 4 ratio, the male as 27 : 6 unit characters in their typical *sex-ensemble*. These numbers are only approximate, and will require modification as our knowledge of this subject increases.

12. THE TYPICAL SEX-ENSEMBLE A PROBABILITY RESULT IN BOTH SEXES.

This probability ratio is well displayed in Coblenz's diagram of the female organs, and in Waldeyer's of the male organs (Plates III. and IV.).

It must now be noted that the characteristic and opposite sex-duct elements in each are inverse as to the extent of their development, the opposite sex-duct elements being at a certain minimum, while the normal sex-ducts are at a maximum. This may be looked on as a probability phenomenon, as in the

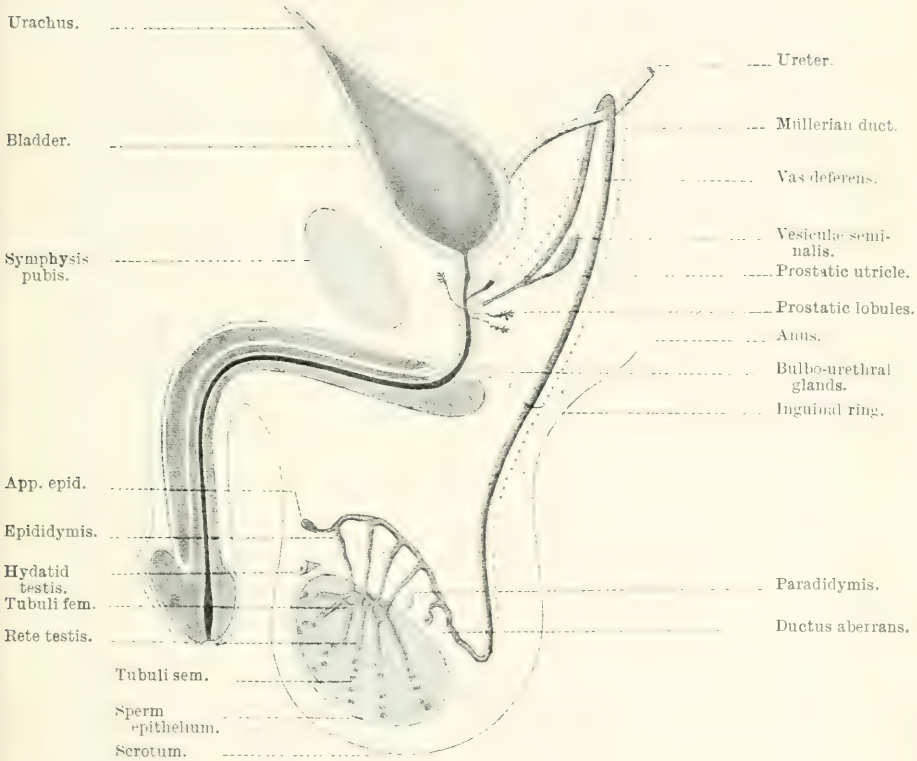

FIG. 1. — Typical male *sex-ensemble* (Waldeyer). *Potent* — Testis, descended; vas deferens, phallus. *Non-potent or opposite sex-duct elements*—Müllerian in dotted line, prostatic utricle. Thus a maximum of potent duct elements and a minimum of opposite sex-duct elements.

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results of coin-tossings. If the heads in ten coins are considered in a mere biometric analogy, as representing the segmented male typical ensemble, the tails in ten coins the female segmented typical ensemble, then, as in a certain number of 10 *coin-tossings we get* 9 *head and one tail or* 9 *tail and one head; so I now go on to show that the result in the typically developed sex-ensemble is a probability one, that there is a maximum of the duct-segments characteristic of the sex, a minimum of those of the opposite sex-duct element, and a sex-congruence in the secondary traits.*

There must be a *minimum* of the opposite sex-duct elements in the human species, otherwise the characteristic elements would be diminished in number as in a 6 — 4 coin-tossing. This disturbed ratio is what gives rise to so-called pseudo-hermaphroditism, as I hope to show in a separate communication. The position of the question is now this. If the unit characters of the genital tract are represented by Weismann's determinants in the heredity cells and their immediate derivative the zygote, and if in the genital tract the potent and non-potent segments have a probability relation, the *locus* of this probability process must be settled, and this is now to be considered.

13. The Probability Result as taking place at the P. G. C. Mass Formation and at Maturation by means of Rearrangement and Losses of the Determinants.

To understand this statement, viz., that the normal urino-genital mammalian tract may be regarded as a probability result, we must now consider the following points.

The unit characters of the urogenital tract (*segmented condition*) *must be regarded in the light of Weismann's causal view already given. The locus of the probability mechanism must be assigned, and the nature of this "tossing" or probability mechanism must be explained.*

On Weismann's theory we must regard the Mendelian unit characters of the adult as represented by determinants in the zygote. As the adult results in the form of unit characters are varied in a probability mechanism and some may be not represented, we naturally ask, Where does this variation and loss take place? What is the *locus* of the change?

The locus of the probability is in the heredity cells and zygotes. In Mendelian crossings with one contrasted character on each side, as, for instance, in the T × D pea plant crossing, it is usually held that the probability result of the segregation of the contrasted unit characters is due to an elementary probability combination of *the pollen grains and egg cells. E.g.*, if a is to be considered as representing the tall character and b the dwarf in a T × D crossing, then $(a+b)^2$ gives $a^2+2ab+b^2$, the probability ratio 1 : 2 : 1, in which the plants with contrasted unit characters segregate *quâ* the latter. This is an exceedingly serious error. The probability result is worked out in *the determinants of the zygote and derived pollen grains and egg cells.* Were it the result of a probability combination of egg cells and pollen grains as such, then, as the action of these is confined to a generation, the probability ratio in the plants (1 : 2 : 1) should come out in F^1. This does not happen, as in F^1 the *soma* of the plant expresses only one contrasted character in this crossing, the tall unit character (*vide* p. 285). What happens really in F^1 is that the determinants for tallness and dwarfness are distributed in a probability ratio (1 : 2 : 1) in the pollen grains and egg cells, while the part of the zygote (oöspore) giving rise to the *soma* of the plant has, *inter alia*, only one contrasted unit character in it (tallness).

The great point is that *we must regard the zygote as the clearing-house for variation allotment of the determinants, the subsequent establishment of related unit characters being the result.* Putting it broadly, relative to the cause of variation, and so far as intrinsic causation at fertilisation and in the zygote are

concerned, we may regard the matter thus. When the zygote is formed there is early set aside a part for the soma of the plant or animal, and a part, the primitive germ-cell mass, for the future heredity cells (primitive germ and sperm cells). A distribution of qualities takes place, and in the primitive germ-cell mass, at anyrate, this follows the law of probability. The results of all adult measurements of "organs" give frequency polygons as a sequence to this.

If a large amount of any determinant is in any special case distributed to the soma there will be less for the primitive germ-cell mass, and this is seen in the progeny of this special soma not equalling it in result. There is thus in heredity not only a transmission but a distribution. In this distribution ordinary continuous variation arises.

In the discontinuous variation (Bateson)—mutation of de Vries—we have a different mechanism, due to losses in the polar bodies, as we shall see. Continuous variation is really an intrinsic property of the germ plasma but cannot be bred for, and variations due to it do not accumulate. The mutation is the striking variation, and its results are transmitted, inasmuch as it is a germ plasma loss.

14. Molecular Physics during the Formation of the Primitive Germ-Cell Mass and in Mitosis and Maturation.

I have attempted to show that the female and male urinogenital tracts, potent and non-potent, are segmental in their nature, that these segments may be assigned from a consideration of the exact deformities of these tracts, and that they may be regarded as unit characters in a Mendelian sense.

I have further urged that the determinants of Weismann in the heredity cells and zygote are causal to them, and that we may regard the distribution of these determinants in the p. g. c. mass and during mitosis and maturation as happening in them. I must, therefore, re-state the phenomena above

mentioned, and then consider, so far as is possible, the molecular physics of the determinants during their active intrinsic changes.

One does not need now to discuss the tremendous influence Dalton's atomic theory had, and still has, on the progress of chemistry and physics. Its more recent expansion by modern observers has given it still greater precision, and to this the researches of physicists on the nature of matter and of electricity, and the effects of electrical discharges in Crookes' tubes and through gases, have, along with radium investigations, largely contributed. The modern chemist and physicist investigate in terms of electrons (Johnstone Stoney), corpuscles (Sir J. J. Thomson), ions (Faraday), and we can now form a clear conception of what happens in an electrical current and in electrolysis of gases and fluids. This line of research and theory has made little impression in pure cytology and embryology, and therefore in this section I wish to make a beginning in what will prove, I have no doubt, a fruitful line of inquiry.

The first great step in biology was made by Weismann when he suggested the fertile idea that we must consider the zygote as containing determinants for the adult organs and the progeny of their possessor. This has been much criticised, especially as it has shaken most seriously the doctrine of natural selection and its necessary sequences, above all, the theory of the transmission of somatic acquired characteristics. I am convinced, however, that time will vindicate the value of Weismann's conception.

As will be seen presently, I look on Weismann's determinants as molecules and ions, the former of which may be neutral when quiescent, the latter positive or negative during mitosis and maturation, and that these two processes are concerned in the production of continuous and discontinuous variation (de Vries' mutation in the latter process).

Thus the phases of mitotic division of a somatic cell and those in maturation or in early zygotes, setting aside of the p. g. c. mass, have a complete likeness, at certain stages of the former, to the magnetic field of the electrician, well seen in the arrangement of iron filings on a conducting plate between the poles of a battery; and in the case of maturation, to the ejection of electrons when an electrical current is passed through a gas. When one, however, tries to apply the former analogy in mitosis, exact explanation fails on the ordinary view of the magnetic field expressed in text-books of physics.

The explanations given by Faraday and Clausius of the electrolysis of solutions of salts such as chloride of sodium or sulphate of copper, and the further work by Sir J. J. Thomson, Larmor, and others on the ionisation of gases, as well as the clearer views on the nature of electricity now held, give us, however, a more exact comprehension of mitotic changes, whether in somatic, heredity, or early zygotic cells, and it is the application of these advances that I now go on to take up.

II.

For clearness and convenience, the Nature of Mitosis, of Maturation, and of the Distribution in the P. G. C. Mass will best be considered under the following heads:—

I. The Changes during the Formation of the P. G. C. Mass and at Mitosis and Maturation.

II. Modern views as to the Nature of Matter, of Electrons and Ions in relation to the Phenomena noted in the preceding heading.

I. The Changes during the Formation of the P. G. C. Mass and at Mitosis and Maturation.

As already explained, when the zygote is formed, a part is set aside to form the somatic part of the future plant or

animal, while the other portion forms the p. g. c. mass for the heredity cells of the individual. There is thus a multiplication and distribution of determinants. The somatic portion gets an Id. of determinants but no so-called sex-determinants. In the p. g. c. mass, which gives the future heredity cells, the determinants are assigned in a probability distribution, as this, we shall see, is the mathematical way we must look on the behaviour of molecules (see p. 304). The measurable adult qualities of progeny are thus, when measured biometrically in sufficient numbers, found to give a frequency polygon.

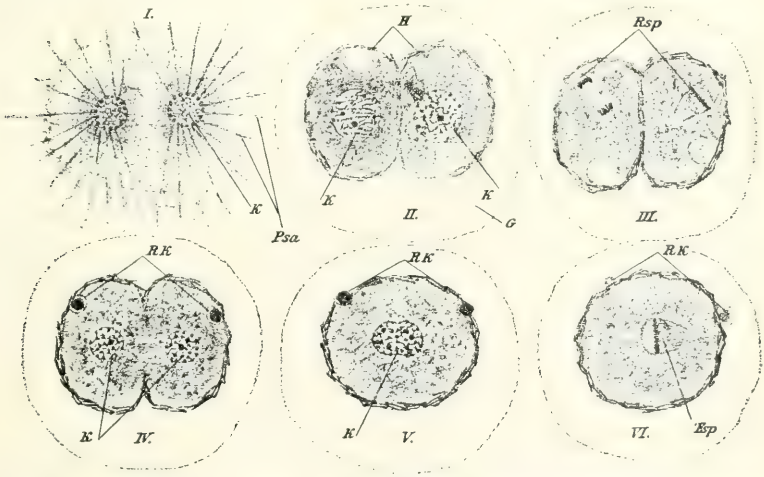
The Structure of a Somatic Cell.—Without going into too much detail, we may consider an ordinary somatic cell as made up of cytoplasm and a nucleus. The nucleus shows under appropriate stains the chromosomes, the number of such varying in different animals.

The chromosomes in a somatic cell contain the causal determinants for similar cells, and it is highly probable that by the mitosis in these, such cells multiply, but such determinants exist only for these cells and cannot change into heredity cells. Their heredity power is thus limited to the reproduction of their like. The idea which has so long unfortunately ruled embryology, that somatic cells can give rise to heredity cells in mammalia, as, for instance, that the germ or capsular epithelium covering the ovary can give rise to its oöcytes, is quite erroneous, and has arrested progress in the mechanism of evolution for many years. It is now being slowly eliminated, and the view of the derivation of the origin of the heredity cells from an early division of the zygote is taking its place. The phylogenesis of the somatic cell is that it is probably derived from the heredity cell, and not *vice versâ* (see *antea*, p. 266).

The Multiplication of Somatic Cells by Mitosis.—When a somatic cell multiplies by mitosis we get a very striking series of changes (Plate V., Fig. A).

A. Partially schematic figure of cell and nuclear division in Ascaris megalocephala (round worm of horse)—Kostanecki and Szymonowicz.

1, Resting cell; 2, Centrosomes after division; 3, Prophase, centrosomes at poles and radiation well marked, chromatin of nucleus in four chromosomes; 4, Mutterstern stage, chromosomes at equator; 5, Metaphase, chromosomes split longitudinally move to the poles; 6, Anaphase, the body of the cell begins to divide; 7, Division of the body-cell nearly finished, nuclei passing into skein stage.



B. From Schaudinn, to show stages in copulation of Actinophrys sol (Ehrenberg).

I. Two free-swimming individuals copulated: *K*, nucleus; *Psa*, pseudopodial axes. II. Beginning of encystment: *G*, jelly envelope; *H*, inner envelope; *K*, nuclei, with skein development. III. In anaphase. IV. *RK*, polar bodies; the reduced nuclei lie already central in their cells; beginning of cell union. V. Nuclear union and polar bodies passing through cell membrane. VI. Beginning of the metaphase, the polar bodies degenerating and losing staining power. (See text, p. 298.)

[*Face page* 296.

Mitosis.—This is a very complicated process, but without going into too much detail we may, in a somatic cell undergoing it, recognise—(1) *prophase*, beginning with a skein condition with two centrosomes. Then comes the achromatic spindle with the skein broken up into distinct segments; finally they form a ring round the equatorial plate. This finishes the prophase. (2) In the metaphase the nucleus divides into similar halves (Flemming) which then separate in (3) anaphase and (4) telophase (see Plate V., Fig. A).

A minute structure known as the centrosome is apparently intimately connected with the mitotic changes, and if we follow current views we get its apparent division into two, accompanied by a rearrangement and division of the chromatin filaments and particles of the chromosomes between these two. At the end of the prophase we get the centrosomes, lateral and radiating lines joining them on each side with the central equatorial plate. The chromosomes then divide longitudinally (metaphase), arrange themselves in two V-shaped and equivalent groups with radiating structural lines connecting them to the centrosome, finally the lines disappear, and we get two complete cells with a centrosome at each lying outside, and a new nucleus with a skein-like arrangement of the chromosomes. From the longitudinal division each derived cell contains the same number of chromosomes as the mother-cell, most probably by a doubling and segregation of the determinants.

It may be urged that as in the final stage each cell may have a centrosome outside, this militates against my view that they are collections of ions, and is in favour of their having a directive power in cell division. It may be, however, that they are really ejected ions giving slight extra variation to cells arising from mitosis, and are thus allied to polar bodies. (See under II. below.)

The Changes in the Heredity Cells at Maturation, i.e., *prior to Fertilisation.*—These are best studied in *ascaris megalocephala*,

as has been done by Boveri and others. We have seen that the number of chromosomes in a somatic cell varies according to the animal or vegetable cell studied, and the same holds good for the heredity cells. In the variety of the ascaris studied they are four in the zygote, and this makes the enumeration of them, in their various changes, easy. From Plate V., Fig. B, the changes in fertilisation of Actinophrys sol are readily seen, and the result is that prior to fertilisation half the chromosomes are thrown off (so-called polar bodies) from each heredity cell (oöcyte and spermatid), and thus by this reduction the union of these gametes makes up for the new zygote the proper number. The reduced oöcyte and spermatid each contains half the chromosomes; they are then termed gametes, while the zygote or fertilised ovum, by the union of two gametes, necessarily contains the proper and full number.

II. Modern views of Ions and Electrons and Matter in relation to the Phenomena described under I. as Mitosis and Maturation.

The view to be advanced, under this heading, as to mitotic and maturation changes is that the former are due to the interchange and temporary persistence of negative and positive ions, that the throwing off of the polar bodies in maturation is an ejection of ions, that the former changes (mitosis) give rise to continuous variation expressed by a frequency polygon, the latter (maturation) by marked variation, the discontinuous variations of Bateson or mutations of de Vries.

The cell ion may be considered as purely electronic or as an exceedingly minute particle of matter — in this case albuminous—electrically charged; and that this material view of its *physical* constitution is a feasible one in the present connection is shown by the staining processes which demonstrate the centrosomes and filaments joining them.

We may regard the filaments and centrosomes as streams and masses of ionised albuminoids (determinants) taking up the special stains employed.

To consider the appearance at Plate V., Fig. 4 A, as a magnetic field does not help, and any comparison of the radiating filaments to Faraday's lines of force, much as this idea has advanced electrical conceptions, gives no clear explanation. Light, however, is thrown on this subject by Faraday's ionisation theory, the result of his electrolysis of fluids, and by the ionisation of gases as described by Sir J. J. Thomson and others. If we combine with this the modern conception of electricity on the electron theory, a clearer view can be formulated as to the mitotic and maturation changes.

Faraday showed that when an electric current is passed through a solution of common salt, sodium is deposited at one pole, the positive, and chlorine given off at the other, the negative. He also proved that the amount of sodium deposited depended on the valency of the metal and the amount of the E.M. force. He formed the fertile theory of ions, the "travellers," and supposed that in the electrolysis of NaCl the electricity atoms combined with the sodium one, and travelled to their appropriate pole. The ion here, therefore, is a charged particle of sodium and electricity in definite proportions.

The view of Grotthuss, held for long, was that the electrical current decomposed the molecules of the electrolyte and that free ions were given off at the poles, the ions between combining temporarily. It was ultimately seen that this was untenable, as the current was not strong enough to decompose water or the chemical solution used in the experiment. A striking and important theory was advanced by Clausius which clears up many difficulties. Clausius holds that in acidulated water undergoing electrolysis the molecules (H_2O)

do not exist as such but as ions, H and OH, constantly forming and combining. There is thus a certain number of ions free, and the electrical current directs these to their appropriate poles. We may look on albumin in the same way. In the chromosomes there are always a certain small number of ions free, and these during mitosis undergo electrolysis, *i.e.* polar direction, in a way to be presently considered.

Constitution of Matter.

We may here note that according to Sir J. J. Thomson we may regard matter as electronic. The electron has the property usually attributed to matter, of mass and inertia, and thus we may exclude the idea of matter as something apart from electrons and regard it as purely electronic. Protoplasm, then, under this view would consist of molecules, certain combinations of electrons, with balanced charges, while in the ions, positive or negative and derived from the molecule, we would have an excess electron or electrons in the negative and one or more fewer in the positive. This gives, therefore, a more simple idea of mitosis. It is under some stimulus that the ions are formed and attract or repel. We may thus regard the protoplasmic molecules and ions as electrically charged matter or as electrons.

Giese, Sir J. J. Thomson, and his followers have extended the view of ionisation to gases with an interesting result on the points discussed.

I may, therefore, here quote M. Lucien Poincaré's clear account on gas ionisation:—"Suppose we pass through some gas, such as hydrogen, at ordinary pressure, a pencil of X-rays. The gas, which till then has behaved as a perfect insulator, suddenly acquires a remarkable conductivity. If into this hydrogen two metallic electrodes in communication with the two poles of a battery are introduced, a current is set up in very special conditions which remind us, when they

are checked by experiments, of the mechanism which allows the passage of electricity in electrolysis, and which is so well represented to us when we picture to ourselves this passage as due to the migration towards the electrodes under the action of the field, of the two sets of ions produced by the spontaneous division of the molecule within the solution" (*op. cit.*, p. 239).

Electrons or Electrical Atoms. — The modern theory of electricity is that it is one, and that what are called positive and negative currents can be explained as follows:—

The result of Crookes's well-known experiment of passing a powerful electric current through a closed glass tube with as perfect a vacuum as possible, is that from the cathode negative atoms of electricity, at one time called radiant matter by Crookes, escape, while positive atoms, not isolated as such, issue from the anode. Crookes's radiant matter consists of electrons or corpuscles negatively charged and formed by the disintegration of neutral atoms.[1]

The negative and positive ions differ in regard to the number of atoms of electricity they contain, the negative having one electron in excess, while the positive ion contains an electron or corpuscle fewer than the negative one. The negative current in electricity has thus an excess corpuscle in its atoms, and therefore electricity is one, not two conditions.

Thus the molecule of protoplasm is neutral, *i.e.*, has in it or is actually made up of balanced positive and negative charges. The electrons are in a state of whirl, and thus electrons are thrown off, giving ions, positive and negative, the former with an electron or electrons less, the latter with an electron or electrons in excess. All these states, molecules, positive and

[1] "The electron is the smallest electrified body capable of separate existence" (F. d'Albe), and repels another electron, the converse of what happens to particles of ordinary matter.

negative ions in relatively smaller numbers, may be considered as being present in protoplasm.

I now go on to suggest that it is the transient development and interchange of such ions that may be regarded as giving the mitosis in developing cells and maturation in approaching fertilising cells.

15. Mitosis and Maturation the Causes of Variation; the one causing Variation without Loss of Determinants, the other Variation by means of their Loss.

Weismann's generalisation that the determinants in the heredity cells and zygote are causal to the developed organs in the adult organism, and the evident corollary that an environmental variation of the body or soma must be rendered causal in the oöcytes and spermatids to be transmitted, has done much to clarify one's ideas as to the cause of variation. No one has as yet given any mechanism by which an extrinsic somatic variation can be made causal; and pangenism, mnemism, etc., have been tried, only to be found wanting.

Weismann's theory of the causality of the determinants in the germ-plasm to the adult structures is based on *a priori* reasoning, but need not remain in a generalised form. We may be able to group such determinants, and ultimately give their relations to one another.

The *sex-ensemble*, typical and atypical, is a probability result, and we are therefore driven to the query, At what period of ontogenetic rearrangement of determinants could such a probability distribution occur? The answer undoubtedly is, at the formation of the p. g. c. sperm- and germ-cell mass. At that time a *sex-ensemble* molecule would be formed containing its electronic determinants. These by maturation at fertilisation and by p. g. c. mass distribution would be in mammals

varied until we got the stable arrangement of the typical *sex-ensemble* molecule, and the varied set for deformities and for atypical sex.

The relation of such molecules to the others in space is more difficult. That there are definite static groupings is suggested by the remarkable cases of inversion of abdominal and thoracic organs, an inversion that may be explained by a supposed half-turn at an early period in static determinant arrangement.

I now go on to suggest that, on Weismann's view given above and the facts and theories under heads I. and II., an intrinsic theory of variation can be formulated which throws light on the subject.

The two processes going on in the chromosomes of the nuclei of the heredity cells, best worked out in the oöcytes, and prior to fertilisation, are mitosis and maturation; in the former, continuous variation takes place in the chromosomes of the heredity cells; in the latter, on each side the chromosomes are reduced to one-half.

It is, therefore, in the pro- and metaphase stage of the mitosis where the centrosomes, one at each side, have the spindle-shaped filaments between them, that we get a real electrolytic picture, the centrosomes being looked on as *each a collection of ions, while the radiating filaments are streams of ions passing to one or other pole, positive or negative.* Thus mitosis may be regarded as an electrical phenomenon, and the various changes can be explained on the theory that the determinants in the chromosomes behave as neutral molecules or as negative or positive ions, and act under the ordinary convention that like repel and unlike attract. The centrosomes, as said before, are accumulations of ions equivalent to the + and − poles. These centrosomes are usually considered as having some directive influence on mitosis, but no explanation has been given as to how they act in this capacity.

In mitosis, therefore, we have an electrolysis, the ions being directed to their appropriate poles and some of them being ejected—the centrosomes—so as to cause variation.

Maturation is an actual ejection of chromosomes at fertilisation, owing to some chemical stimulus to be discussed later on. If, then, the determinants in mitosis act in this manner, it may be asked, Can their movements be described under any mathematical formula? Supposing one could observe the rates of movement, distances travelled, impact, and distributions in amount made in the molecules during mitosis and maturation, is there any generalisation that would enable us to understand the probability polygons by which the sizes and functions of the organs in the adult organism are expressed? For this we must consider the behaviour of gases in a closed space and the emission of particles from radium.

In a closed vessel filled with gas at ordinary temperature and pressure, the pressure on the walls is due to the impact of the molecules. The speed of these varies, but according to Clerk Maxwell we can regard the amount of their various impacts as expressible by a frequency polygon.

On this point M. Lucien Poincaré remarks (*op. cit.*, p. 100):—"It was Maxwell who first thought of introducing into the kinetic theory the calculation of probabilities. Willard Gibbs and Boltzmann later on developed this idea and have founded a statistical method which does not, perhaps, give absolute certainty, but which is certainly most interesting and curious. Molecules are grouped in such a way that those belonging to the same group may be considered as having the same state of movement; then an examination is made of the number of molecules in each group, and what are the changes in this number from one moment to another. It is thus often possible to determine the part which the different groups have in the total properties of the system and in the phenomena which may occur."

A similar condition is also supposed by Arrhenius to exist in the emission of particles from radium. "The number of α-particles emitted by a solid radio-active substance in unit time is not constant, but changes according to the theory of probability."

We thus see that molecules and ions may be considered as having their movements and distribution recorded by a frequency polygon, and thus the frequency polygon in measurement of adult organs given by biometry.

The question may be summed up as follows:—

Mitosis is the result of the movements of ions, *i.e.* electrified albuminoid determinants. In the single skein arrangement we have neutral molecules moving under Brownian movement, and on this Reuss says that small particles in a fluid are moved by an electric current. This indicates that they carry a certain electric charge. The surrounding fluid is charged with the opposite electricity, and is therefore carried in the opposite direction by the current.

Brownian movement may thus be regarded as due to an electrical reaction between the fluid and the particles, whereas in mitosis and maturation the reaction is between the ions.

When we have double filaments in mitosis these are charged with like electricity, and therefore are repelled, *i.e.* separate, and necessarily equal in size to the eye. The same holds good for particulate conditions of the chromosome. In the spindle arrangement between the centrosomes, which I suppose to be collections of ions, and the filaments of the spindle, ions travelling to the centrosomes, we get electrolysis going on, *i.e.* separation and segregation of groups of determinants for redistribution.

Maturation.—If we take Sir J. J. Thomson's corpuscular theory of the atom of matter as consisting "of a large number of corpuscles" (electrons) "all electrified negatively and held together by positive electricity equivalent in amount to the

sum of the negative charges of all the corpuscles so as to produce an electrically neutral atom" (Mellor, *op. cit.*, p. 838), and if we add to it what he supposes to happen during the ionisation of gases, viz., "that the normal process of gaseous ionisation consists in the detachment from an atom of the gas of a minute particle called a corpuscle" (Whetham, *op. cit.*, p. 156), one may regard the throwing off of the polar bodies as an ejection of ions on the part of the approaching spermatid and oöcyte, making them gametes, until a condition of opposite charge occurs; we then get attraction between them, *i.e.* fertilisation.

In all the mitotic and maturation charges we get a microscopical representation of what happens in the electrical charges used by the physicist when electrolysing fluids or gases.

As the gamete and zygote heredity cells form a cycle, we may now summarise as follows:—

As already explained, the zygote has early set apart in it a portion, termed the primitive germ-cell mass; from this the heredity cells (primitive germ and sperm cells) arise, and these give rise in the sex glands to the oöcytes and spermatids. By maturation the heredity cells become gametes and the zygote arises from their union.

What is to be specially studied under this head is the question of what happens when the formation of the primitive germ-cell mass is followed by that of the heredity or primitive germ and sperm cells. In the human embryo the primitive germ cells have been found in the endoderm, travelling towards the Wolffian ridge (Ingalls). The heredity cells are due to a multiplication and probability distribution of the determinants of the zygote, therefore a distribution of these by the law of probability among the heredity cells. The one set apart for the soma of the adult the zygote gives rise to, receives afterwards in its Wolffian ridge the heredity cells whose origin has just been given from the p. g. c. mass, and thus the entire

individual expresses in its *soma* the result of one Id.[1] of determinants and in the *sex glands* many thousands of divided Ids. (see p. 266), each oöcyte and spermatid containing an Id. or fraction of an Id., but lacking the power of developing somatically. As all these processes happen according to a probability law, variation in the determinants of the heredity cells is the result, and we thus get an explanation of the well-known biometric fact that measurements of organs[2] in the adult give a frequency polygon. This shows the far-reaching nature of the work of biometricians.

Thus in the heredity or primitive germ cells we have a probability distribution of all the multiplied determinants of the zygote, excepting that part set aside for the "soma" of the plant or animal, which, of course, has also had its probability Id. allotted to it. Thus arises the variation of progeny and also another important consideration. Children always vary from their parents, and the parents do not transmit some of the qualities they possess. Genius, for instance, is not again distributed. A simple deduction from what has gone before helps us to understand this. We may suppose that in the zygote giving rise to a genius there has been an unusually large distribution of those cerebral determinants connected with genius, and thus the great poet, artist, or scientist. There would, therefore, as a corollary, be less of such determinants for the probability distribution in the primitive germ-cell mass giving rise to the heredity cells in this man of genius. In heredity, there is not only transmission but also distribution, and therefore the extra amount put into the somatic Id. of the zygote in a genius leaves less for his p. g. c. mass, and therefore for his progeny. The determinants in all the Ids. in an adult

[1] Whether the Id. of the heredity cell is equivalent to that of the somatic part of the zygote is not yet settled. Probably the somatic Id. has something in it, as yet unknown, determining its somatic development.

[2] The measurable elements of an adult organism.

(somatic and propagative) vary, and the chance of equivalent Ids. in single heredity cells is so minimal as to be yet unknown. The chances of the child being the same as its parent in every respect are negligible; parents give rise to a variation progeny, a fact of a disappointing nature to many anxious and able fathers and mothers.

Mitosis, maturation, and the formation of the heredity cells are variation phenomena, give results summed up by the law of probability, and are in their action governed by the electrical mechanisms so intimately bound up with molecular distribution.

Action of Internal Secretions.—Finally, an interesting question arises as to the possibility of internal secretions inducing mitosis, *i.e.* setting up electrical attractions and repulsions by their stimulus. From what has gone before it may be a purely physical action; that is, an internal secretion so stimulates the neutral molecules that by the loss of electrical atoms owing to their increased whirl they became positive and negative ions and thus we get the attractions, repulsions constituting mitosis, in sperm and germ cells the ejection of masses of ions constituting maturation. Beard made the very interesting observation, without comment, that when the primitive germ cells of elasmobranch embryos were travelling through the germ path (Keimbahn) their nuclei were not undergoing mitotic changes. This goes on, however, markedly when they pass into the genital ridge, and presumably it may be an internal secretion, afterwards the ovarian secretion or hormone, which is the cause of the interesting mitoses of the oöcytes there.

We now go on to apply the previous statements to the question of the *sex-ensemble.*

16. Sex Phylogenesis and that of the Sex-ensemble must be studied to explain the Origin of the Heredity Cells and Derived Gametes and that of the Duct portion of the Genital Tract.

In now summing up the evolution of the *sex-ensemble* we have to recall the phylogenesis of the heredity cells (primitive germ and sperm cells) giving rise to the gametes as well as the phylogenesis of the duct elements in the urogenital tract (Wolffian and Müllerian duct derivatives); also the phylogenesis so far as known of the urinogenital sinus and external genitals.

Of these the phylogenesis of the heredity cells and gametes is by far the most ancient, going well back into fossil times. The duct elements have a much shorter ancestral line, reaching only to the organisms with nephridia (annelides), although these, of course, have their forerunners, but these duct elements are mere parvenus compared with the heredity cells.

We have thus to consider (*a*) the phylogenesis of the heredity cells and gametes; (*b*) the phylogeny of the pronephros and mesonephros and of their ducts.

(a) *The Phylogenesis of the Heredity Cells and Gametes.*—The special question that arises under this is what view one has to take of an elementary unicellular protozoon like the amœba, such, for instance, as the Amœba crystalligera (see p. 266 for zygotic origin of heredity cells). We may regard it as an organism equivalent to a primitive germ or sperm cell—a heredity cell—motile or non-motile and placed in an external environment *i.e.* outside the soma of an organism.

While, at first sight, an extremely simple cell, it has by no means an elementary organisation, but performs a great number of functions and is of a most complex chemical constitution. According to the opinion of a friend, an expert in physiological chemistry, we may regard living protoplasm as "an aggregate of highly complex organic substances of a

colloidal nature (such as proteins and lipoids and other substances of unknown chemical constitution, which have the power to act as catalysts), and of inorganic electrolytes which are partly kept in a state of absorption by the colloids, the whole of which is surrounded by a semi-permeable membrane constantly undergoing chemical changes."

It can perform all the functions of the highest organisms, viz., reproduction, metabolism, phagocytosis, excretion, formation of internal secretions, of powerful irritants or toxins in higher but still elementary organisms, and possesses power of locomotion, injection, ejection of excreta.

Even specialisation of function has begun, as "in a flagellate protozoon called euglena a pigment spot exits near the anterior end," probably an eye spot (Washburn, *The Animal Mind*, p. 122). It has perhaps an elementary mind. Its physical structure may be regarded as made up of neutral molecules with positive and negative ions, and either as purely electronic or as electrically charged protoplasm, *i.e.* protoplasmic and electronic.

It is thus quite a misnomer, therefore, to speak of the "simple" protozoon. It is rather a profound and elemental complex, and has the greatest mystery of all phenomena—life.

Its reproduction may take place by amitosis (F. E. Schultze and Schaudinn) and also by mitosis. The former Schaudinn speaks of as being demonstrated "ausser Zweifel." He made some of his observations on Amœba crystalligera, and found there direct division going on, as F. E. Schultze had already demonstrated in A. polypodia.

In A. binucleata Schaudinn found mitosis taking place, and figures the stages of spindle formation.

An important section of Schaudinn's work is in relation to the copulation of Actinophrys sol (Ehrbrg.). In this, as Plate V., Fig. B, shows, we have nearly all the mitotic and maturation changes described, in the maturation and fertilisation

changes of higher organisms, *i.e.* in Ascaris megalocephala. We can note the junction of two equivalent amœbæ—(1) their encystment and double condition; (2) with the chromosomes in the skein condition (Knäuelbildung); in (3) the formation of the prophase stage; while in (4), (5), and (6) we see the formation of the polar bodies and their ejection.

This is equivalent to a real fertilisation, leads to a variation in two similar organisms, and shows us that basally the aim of fertilisation is "variation." The determinants in the chromosomes become varied, and the ejection of the polar bodies, a "mutation," gives a more marked variation. The Amœbæ sol engaged in copulation here are similar organisms, and therefore "sex" is not present.

The succeeding stages in the development of sex may be hypothetically sketched as follows:—The copulation of a non-motile and flagellate protozoon would be the next important stage, and this would begin sex, as they would give motile and non-motile gametes. When such protozoa formed colonies we might then get the penetration of ciliated and non-ciliated organisms into such a colony, and they might be fixed in some certain situation by a "hormone." This would lead to the formation of a hermaphrodite sex gland, and as the heredity cells would be derived from the ciliated and non-ciliated heredity cells in the indifferent sex gland, a hermaphrodite condition would arise in the organism, *i.e.* it would have ciliated and non-ciliated heredity cells.

As one passes up the scale of life the hermaphrodite condition would be eliminated. Even in organisms where this is actually present, it falls into abeyance, but altimately the most stable and effective condition would be that where one organism, male, had motile heredity cells produced in abundance, and necessarily in tubuli, which when shed went in search of the egg produced in a follicle. Hermaphroditism leads to inbreeding and degeneration from a small range of variations, whereas

sexual reproduction, from its greater power of variation, would give stronger offspring. The condition of certain abnormal fishes and frogs where a hermaphrodite condition frequently exists, shows the process of elimination of hermaphroditism still lingering. In the mammalia, finally, all hermaphroditism is gone, but there is no reason, theoretically, why heredity cells, male and female, might not appear in a sex gland. This has never been satisfactorily demonstrated, as all alleged ova in the so-called ova-testis in mammals are probably isolated sections of tubuli seminiferi in a defective male. The organ is really a defective testis.

The heredity cells in mammals are thus descended from elementary protozoon organisms which have become embedded in another organism, and under the influence of hormones, in special sex glands. The gametes are derived from the heredity cells by ejection under the same molecular and electrical processes seen in the ionisation of gases.

(b) *The Phylogeny of the Pronephros and Mesonephros and of their Ducts; Origin of the Opposite Sex-Duct Elements.*—In the human mammal the Wolffian bodies are prominent in fœtal life only up to the second month. They are of the greatest importance, not only because of their excretory function, although this is denied by some, but also from the fact that the sex glands develop on them and in the male, part of the tubules become tubuli seminiferi, while the Wolffian duct becomes the vas deferens.[1] The female potent tract arises from the Müllerian ducts mainly. In the female the parts giving rise to the male epididymis form the degenerated epoöphoron with its short duct, and the paroöphoron, the opposite sex-duct element, but the lower end becomes the hymen, a functional structure. In the male the opposite sex-duct elements are the hydatid testis and the prostatic utricle. *The evolution of the duct portion of the urogenital tract of*

[1] The development of the testis still requires much elucidation.

mammalia is, in the female, a progression from a complex condition to a simple one, and the object of what follows is to show how this change takes place. Briefly, it may be summed up as follows:—

It has been already shown that in the human female we have in the urinogenital tract exact losses leading to deformities, or, in a broad sense, to variations. In a typical *sex-ensemble* we have an arrangement of the duct portion characteristic of the sex, and also an opposite sex-duct portion degenerated and imperfect. This I suppose to be due to its losses in the past at maturation while in the determinant stage, when the polar bodies are thrown off, and thus a probability and working arrangement is brought about.

The urogenital system has autonomous determinants for it in the somatic part of the zygote; these are expressed in the typical tract by unit characters or segments, and thus we can reckon out how many unit characters make up the system and what polar losses lead to the ultimate ratio between the potent and opposite sex-duct elements in typical sex.

We must, therefore, first attempt to fix what might be the possible segments in the potent and opposite sex-duct structures in a typical human mammal, basing this on the results of the exact losses in the atypical urogenital tract. This has been already done in the typical human genital tract, and I have taken them tentatively as follows:—Human female genital tract, potent, 40; non-potent, 4. In the male it is—potent, 27; non-potent, 6 (*vide* p. 288).

17. Number of Unit Characters in Lower Mammals.

If we now go back to the lowest mammals—the monotremes—we find an unsatisfactory state of knowledge as to the urogenital tract, especially as to the opposite sex-duct elements, and Professor Anderson Stuart of Sydney tells me that this

element has not as yet been worked out by Australian anatomists. I therefore go on to the—

Marsupialia.—In the marsupialia I examined the genital tract in the female rat-kangaroo microscopically and serially, and found, *inter alia*, the epoöphoron well developed. The genital tract in the female rat-kangaroo is complex and made up of the following elements:—*Potent*—Ovary, 2; tube, 6; bicornuous uterus, 4; Müllerian portion of vagina, usually blind below, 2; urinogenital sinus, 2; Wolffian portion of vagina (lateral canals), 2. *Non-potent*—Epoöphoron, 2.

This gives 18:2 as the potent and non-potent ratio, but I do not insist on it, as much work remains to be done here.

The other orders of the mammalia should now be considered relative to their *sex-ensemble*, so that they might be compared with that of the normal human male and female. This, however, cannot be done at present, chiefly owing to our deficient knowledge of the opposite sex-duct elements present, but it is to be hoped that in time the innumerable gaps in our knowledge will be accurately filled up. There is, however, the same ratio tendency in these, but the potent are fewer in number, the non-potent greater, with the preponderance in number of segments always greater on the potent side. If the human female genital potent and non-potent ratio is 40 potent to 4 non-potent, then in the other members of the mammalian phyla we may expect a 39:5, 38:6, 37:7, etc., ratio of potent and non-potent organs, and thus establish a ratio table to be filled up by future investigators. The same may be found in the male genital tract, but our ignorance on these points in this sex is colossal.

18. Summary.

Sex is established early in phylogeny by the formation of gametes from primitive germ and sperm cells, i.e., *flagellate and*

non-flagellate, protozoa outside an organism ; then follows their lodgment in an organism, and ultimately in its sex gland.

The human potent genital-duct tract and the opposite sex-duct elements constituting with other structures the typical sex-ensemble, *have a maximum-minimum,* i.e., *probability relation, due to successive polar body losses of the autonomous determinants causal to adult results at maturation, and are recorded in the germ plasma* (Mendel).

Natural selection eliminates ineffectual results, and thus a stable condition of the sex-ensemble *is reached.*

Under this heading must be summed up the views as to the nature and determination of sex, and the *sex-ensemble* of which the foundations have been laid in the preceding pages.

Sex is regarded as due to the nature of the heredity cells. Any metazoon with motile heredity cells is male; with non-motile ones it is female.

One must distinguish between *sex* and *sex-ensemble*. The latter has not only the sex gland but the genital ducts according to the sex, *i.e.*, the Müllerian ducts, part of the urinogenital sinus, the external genitals and secondary sex characters, viz., fully developed mammæ, special type of pelvis, hair distribution, etc., in the female ; while in the male there is the testis as sex gland (descended), the vas deferens, the prostatic utricle, external genitals with the secondary sex characters, mammæ (rudimentary), pelvis, hair distribution. Further, as part of the normal *sex-ensemble* we have a minimum of opposite sex-duct elements, a most necessary feature of the normal or typical *sex-ensemble*, inasmuch as any increase of this minimum necessarily has a diminution of the potent and characteristic tract, and therefore an atypical form of sex arises—what is usually but erroneously described as pseudo-hermaphroditism.

We may regard the typical protozoon as a primitive germ or sperm cell, leading an independent existence, as made up of protoplasmic molecules with some free ions, and these, as

already described, are to be considered as made up of electrons and, according to their electrical condition, as neutral molecules and positive and negative ions. In the first the positive and negative charges are balanced, while in the negative ions there is an excess electron (or atom of electricity), and in the positive a loss of an electron. The neutral molecules have, however, a constant whirl in them, and thus by loss of electrons become ions.

We may leave out the question of the significance of mitosis in protozoa and take up the behaviour of the protozoon when its molecules are negatively and positively charged and are regarded as ions. In the electrical whirl of the electrons of the molecules we get an ejection of electrons as in ionised gases, and thus the formation of gametes by the loss of the polar bodies in the approaching heredity cells.

In Actinophrys sol (Plate V., Fig. B) we have a copulation of similar organisms and all the phenomena of fertilisation. This copulation is for the production of variation, and is the first step in the production of sex. We may then imagine the union of a ciliated and non-ciliated protozoon as the next stage. The organism this gave rise to would in like manner form ciliated and non-ciliated gametes.

When such a hermaphrodite protozoon entered a colony of protozoa and became locally fixed, the sex gland would be established, and necessarily with both sperm and germ cells—a true hermaphrodite condition.

In such organisms it is known that self-fertilisation gives rise to degeneration and fertilisation is ineffective. In flowers, for instance, self-fertilisation may be prevented by local arrangements or by difference of ripening of the male and female elements. Natural selection would then act and cross-fertilisation by insects become effectual. There would thus be a tendency for the heredity cells in an organism to become of one kind—male or female. The hypothetical sketch of the

origin of primitive sex by the passage of hermaphrodite protozoa into a protozoon colony and their establishment locally and ultimately in a higher organism as a sex gland is consonant with Haeckel's law.

In higher animals, fish, amphibia, mammals among others, the heredity cells formed by an early division of the zygote pass through the developing somatic part of the zygote and establish themselves in the genital ridge of the Wolffian body to form the sex gland. The heredity cells passing in would in far-back organisms be of both kinds—male and female; and in so-called hermaphrodite frogs and fishes we have instances of imperfect previous elimination. From all this it follows that sex, defined by the nature of the heredity cells, arises far back in phylogeny, and is produced by the molecular reduction of these under special electrical conditions in two kinds of protozoon organisms characterised by the contrasted conditions of activity and passivity—on which Geddes and Thomson base so much under the terms anabolism and katabolism.

The question of the establishment of the potent sex duct and opposite sex duct or non-potent elements in mammalia must now be summed up. This is an important element in the urogenital system, and while sex cannot be defined from their existence the *sex-ensemble* is not typical unless the non-potent is present in a minimal condition. In the typical *sex-ensemble* the opposite sex-duct element must only be represented to such an extent that it does not deprive the potent part of the tract of segments necessary for the exercise of its full functions. What we know of its exact amount is still far from complete, but the following sketch of the potent and non-potent elements, so far as it is yet known, is as follows:—In the mammalia we find in *marsupials* the bicornuous uterus and a remarkable condition of the vaginal tract. In it we have the lateral canals well developed, opening above into the Müllerian vagina and below into the urinogenital sinus. The lining of each is a

multiple squamous epithelium like that of the human vagina, and these lateral canals, which are, I believe, the Wolffian ducts, probably conduct the spermatozoa into the upper part of the Müllerian vagina, and thus to the uterus.

The central portion of the vaginal tract is usually a pouch, blind below but open above. This central portion is lined with a single layer of columnar epithelium. In some species the central pouch opens below into the urinogenital sinus (Bennet's kangaroo). Sexual congress is thus accomplished, so far as the female is concerned, by intromission of the phallus with the urinogenital sinus, but in Bennet's kangaroo the central or Müllerian portion is also concerned.

Whether in the cases of the kangaroos with the blind Müllerian pouch, birth of the small fœtus is accomplished through the lateral canals, or whether the blind portion yields at its tip so that the fœtus passes from it into the sinus (J. P. Hill), is not yet determined. The vagina of the *human female* is Müllerian in its upper two-thirds, the lower third being derived from the urinogenital sinus, and thus in the kangaroo these two elements are separated and not blended as in the human female. As we pass up the mammalia we get the Wolffian duct element more or less eliminated, and thus the human vagina becomes a single canal with the Wolffian duct element much less represented. In the cow and sow it is represented in the vaginal tract by a narrow tube (Gartner's canal); this is less represented in sheep (Numan), but in other mammals its exact representation is almost completely unknown.

From what has been said of the segmental condition of the urogenital tract and of the significance of the polar losses, we may regard the human genital-duct tract as the result of a series of polar body losses in lower mammals until an ultimate probability result giving a maximum of potent and a minimum probability result ensues. Any increase of the latter would

lead to a diminution of the potent organs, what lies at the root of the question of atypical sex (pseudo-hæmaphroditism).[1]

From what has been said as to the essentials of Mendelism, it is evident that in the establishment of a variation by a maturation loss, pure in the heredity cells while other cells exist which are not pure to this loss, we get ultimately a pure result in certain cases, and the occasional occurrence of undistributed results giving older forms of the tract.

For the many hundred theories of sex Thomson and Geddes and Beard may be consulted.

Recently special attempts based on Mendel's theories of dominance and recession have been made by eminent observers to settle the origin of sex at fertilisation. Dominance and recession, however, merely express too strongly the fact that in an ordinary plant crossing where two contrasted qualities are present, one is alone expressed in F^1, *i.e.* it is the first of the two qualities to be expressed in the plant soma. In succeeding generations both contrasted qualities are expressed in a probability ratio in the plants because their determinants have been distributed in a probability ratio in the pollen grains and egg cells of F^1. Dominance and recession do not, therefore, apply after the F^1 plants, and have not the significance usually attached to them. Indeed a careful study of the results obtained as to sex by these investigations leaves one in the position of the scholastic student of old who plaintively said of his arid studies—

> "Myself when young did eagerly frequent
> Doctor and saint, and heard great argument
> About it and about; but evermore
> Came out by the same door wherein I went."

[1] This passage from a complex condition to a simple one by successive losses is illustrated in many other conditions in mammals, especially in the fossil predecessors of the horse, when the multiple digits in the legs are eliminated, and ultimately represented in the modern horse by the single bones well known to all.

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ON THE ATYPICAL MALE AND FEMALE *SEX-ENSEMBLE* (SO-CALLED HERMAPHRODITISM AND PSEUDO-HERMAPHRODITISM).

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"*Masculus intravit, fonte emersit utrumque,*
Pars est una patris, cætera matris habit."
Martial.

The belief in the occasional occurrence of hermaphroditism [1] in the human race, although present in all ages, dates practically from Ovid's fable of Hermaphroditus, the son of Hermes and Aphrodite, with whom the nymph Salmăcis fell in love so desperately that she prayed the gods to unite them in one person. This was accomplished in the fountain, as Martial has celebrated.

This poetical and transcendental idea has so dominated the lay and scientific mind that even now the question of hermaphroditism in the mammalia is discussed and supported by the description of specimens with alleged ova and spermatozoa in a common sex gland, or of those where the opposite sex-duct element is unduly represented at the expense, we shall see, of the potent normal tract. It may be noted *in limine* that the existence of the only possible form of hermaphroditism,

[1] It would be better to adopt the French terms Hermaphrodisme and Pseudhermaphrodisme. Those used here are clumsy in comparison, and the French ones are etymologically quite correct. *Sexe-ensemble* is preferable to *sex-ensemble*, as the latter is really an English-French compound.

the so-called glandular one, where an ovotestis is present, has never been demonstrated although theoretically possible.

Pseudo-hermaphroditism is constantly used as a descriptive term, but we cannot have a pseudo form of a non-existent condition, and it is therefore merely a convenient but misleading term for certain cases of atypical *sex-ensemble*, and is based on an erroneous idea of what constitutes sex.

The Criterion of Sex.

The only criterion of sex is the sex gland. Abnormal development of the opposite sex-duct elements cannot be admitted as evidence of sex at all, although they have their importance as fully determining the *sex-ensemble*, typical or atypical.

In typical and atypical *sex-ensemble* cases (hermaphroditism and pseudo-hermaphroditism) the sex is thus either male or female.

Sex-ensemble.

What is concerned in discussing this portion is the "*sex-ensemble*," the "altogether" of *efficient* sex. We thus speak of—

(1) *The typical female sex-ensemble.*—This is made up of (*a*) the ovary; (*b*) the *potent sex-duct tract*—tubes, uterus, vagina, and external genitals; (*c*) the *opposite sex-duct element*—epoöphoron, degenerated equivalent of the epididymis of the male; (*d*) the *secondary and congruent sexual characters*—hair distribution, pelvis, body form, vocal cords, ossification of thyroid cartilages (incomplete), and the psycho-sexual feeling for the male; mentality less strong than in male.

The typical male sex-ensemble comprises (*a*) descended testes; (*b*) vas deferens and phallus—the *potent organs;* (*c*) *the opposite sex-duct elements*—hydatid testis and prostatic

utricle; (*d*) the *secondary sexual charactres*—hair distribution, pelvis, body form, vocal cords, ossification of thyroid cartilages (complete); and the psycho-sexual feeling for the female; mentality stronger than in female.

A crucial point is this: In the *typical female sex-ensemble* we must have the ovary normal, the sex-duct elements (tubes, uterus, and genitals) at a maximum, the opposite sex-duct elements at a minimum, and the secondary sexual characters congruent with the sex.

On the other hand, *the typical male sex-ensemble* must have the descended testes normal, the sex-duct elements at a maximum, the opposite sex-duct elements at a minimum, and the secondary sexual characters congruent with the sex.

It is the disturbance of such relations that constitutes atypical male and female sex-ensemble—i.e., in them the sex glands are recognisable but often aplasic; the characteristic sex-duct elements are diminished, the opposite sex-duct elements increased, and the secondary sexual characters not in every respect congruent with the sex.

Classification.

It must be noted that in classifying atypical female and male *sex-ensemble* (pseudo-hermaphroditism) we base it only on those cases where the question of typical or atypical *sex-ensemble* arises; that is, we do not admit abnormalities not affecting the question of sex. The non-recognition of this has led former authorities to include many irrevelant conditions.

The chief modern classifications have been by Klebs, J. Y. Simpson, Neugebauer, Menge, and a short account of these must be given before the question of one based on recent work and my own is taken up.

Sir J. Y. Simpson's classification is as follows:—

- Hermaphroditism.
  - Spurious
    - In the female
      - From excessive development of the clitoris, etc.
      - From prolapsus of the uterus.
    - In the male
      - From extroversion of the urinary bladder.
      - From adhesion of the penis to the scrotum.
      - Prom hypospadic fissure of the urethra, etc.
  - True
    - Lateral
      - Testis on the right and ovary on the left side.
      - Testis on the left and ovary on the right side.
    - Transverse
      - External sexual organs female, internal male.
      - External sexual organs male, internal female.
    - Vertical or double
      - Ovaries and an imperfect uterus with male vesiculæ seminales and rudiments of vasa deferentia.
      - Testicles, vasa deferentia and vesiculæ seminales with an imperfect female uterus and its appendages.
      - Ovaries and testicles co-existing on one or both sides.

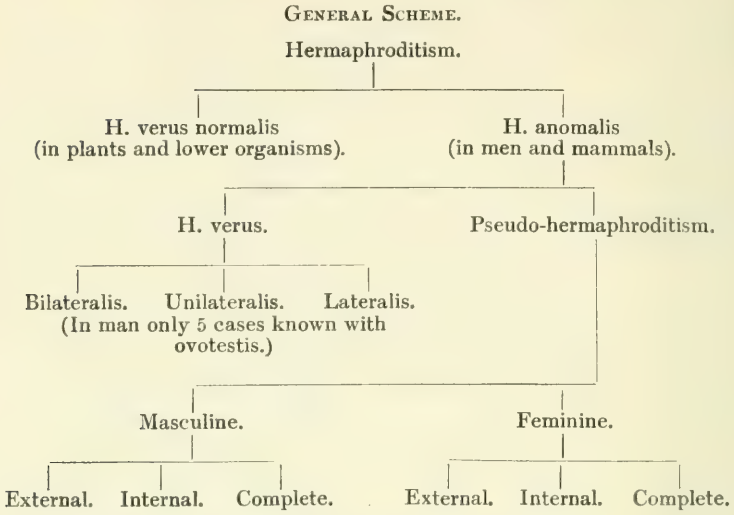
It is evident that this classification, excellent as it was at the time Simpson wrote, included many instances where no question of doubtful sex exists. Lateral and transverse conditions are based on an erroneous opinion as to what constitutes sex. The co-existence in the same individual of an ovary and testis has never been demonstrated in mammals.

Klebs' older table is as follows:—

- I. Pseudohermaphroditismus feminismus.
  - *A.* Internus.
  - *B.* Externus.
  - *C.* Completus.
- II. Pseudohermaphroditismus masculinus.
  - *A.* Internus.
  - *B.* Externus.
  - *C.* Completus.

This is a simpler classification, but is misleading in making other factors enter into the question of sex instead of the nature of the sex glands alone.

Neugebauer gives an excellent and comprehensive classification, but follows Klebs' in its errors:—



Menge's may be given as a modern classification :—

I. Pseudohermaphroditismus masculinus.
II. Pseudohermaphroditismus femineus.

I. { *a*. Glandularis. *b*. Tuboglandularis. *c*. Tubularis.

II. { *a*. Glandularis. *b*. Glandulotubularis. *c*. Tubularis.

The exclusion of hermaphroditism is an improvement. The division glandularis has not been demonstrated, and the tubular and glandulotubular subdivisions are misleading.

What we are really discussing at present is not atypical sex but atypical *sex-ensemble*. Sex is not atypical in any case, but either male or female, and it is the *sex-ensemble* as already defined that is atypical in what are called pseudo-hermaphrodites.

If one admits in evidence in judging the existence of sex, male or female, the normal duct-element and the opposite sex-duct elements, then the normal human male and female are pseudo-hermaphrodites, inasmuch as each has opposite sex-

duct elements, the epoöphoron in the female, the hydatid testis and prostatic utricle in the male.

I have already shown in a previous paper (page 263) that the potent and non-potent segments in the genital tract (sex glands excepted) are to be considered as made up of Mendelian unit characters causally represented in the zygote by autonomous determinants, and that the relation of these potent and non-potent segments in each *sex-ensemble*, so far as the duct elements is concerned, is a probability result, the potent being at a maximum, the non-potent at a minimum, with the secondary sexual characters congruent. The losses of segments leading to this ratio are supposed to take place at fertilisation, and to be due to polar losses at maturation, as already explained in the paper mentioned above.

In that paper I have enumerated the segments in the typical male and female *sex-ensemble* as follows, but I must amplify the former enumeration of the unit characters by adding to the female, Skene's ducts (2), and to the male, the prostate (3). The study of atypical *sex-ensemble* has shown me this clearly, and some segments must also be added for the secondary sexual characters.

Female Sex-ensemble with Enumeration of Unit Characters.

Potent.—Ovaries (2); Fallopian tubes (6); uterus (body and cervix) (4); vagina, upper two-thirds (2); vagina (from urinogenital sinus) (1); hymen (2); vestibule (1); larynx (1); suprarenals (4); round ligaments (2); ovarian ligaments (2); broad ligaments (2); glans clitoridis (2); prepuce (1); C. cavernosa (2); absorbing factors as to cervix, uterus, vagina, hymen (4); Skene's ducts (2).

Non-potent.—Epoöphoron and ducts (4). Thus potent to non-potent 40 : 4.

The Secondary Sexual Characters do not lend themselves as yet to such an enumeration.

Male Sex-ensemble with Enumeration of Unit Characters.

Potent.—Testes (2); epididymes (2); vasa deferentia (6); vesiculæ seminales (4); prepuce (2); C. spongiosum (1); C. cavernosa (2); gubernacula (4); prostate (3); suprarenals (4); larynx (1); absorbing factors in scrotum (1).

Non-potent.—Hydatid testis (2); prostatic utricle (2). Thus the potent to non-potent ratio is 32 : 4 in the typical male *sex-ensemble.*

In addition there are the secondary sexual characters; these do not lend themselves at present to such enumeration, but we may say that both in the male and female *sex-ensemble* they must be congruent with the sex.

These give for typical *sex-ensemble*, a sex gland, a ratio of maximum and minimum between the potent and non-potent, and a distribution of the secondary sexual characters congruent with the sex.

I now go on to show that in atypical female and male *sex-ensemble* we have (1) a sufficiently characteristic sex gland, male or female; (2) an increase of the minimum at the expense of the maximum; (3) a non-congruence of the secondary sexual characteristics with the sex. I shall afterwards attempt to show that in each atypical *sex-ensemble* the result is a probability one in relation to the various segments making up the tract.

Author's Classification.

I. *Atypical Female Sex-ensemble.*—Sex glands female; opposite sex-duct elements in varying amount but not in minimum of the typical *sex-ensemble;* potent segments present but not in the maximum of the typical *sex-ensemble;* secondary sexual characteristics in some degree non-congruent.

II. *Atypical Male Sex-ensemble.*—Sex glands, testes, undescended or descended; opposite sex-duct elements in varying states of representation, but not at the minimum of the typical

FIG. 1.—Atypical female *sex-ensemble:* Anterior view of genitals in Fibiger's Case 2. Note the apparent male hypospadias. The fundus uteri, tubes, and ovaries are seen above the pubes (Fibiger).

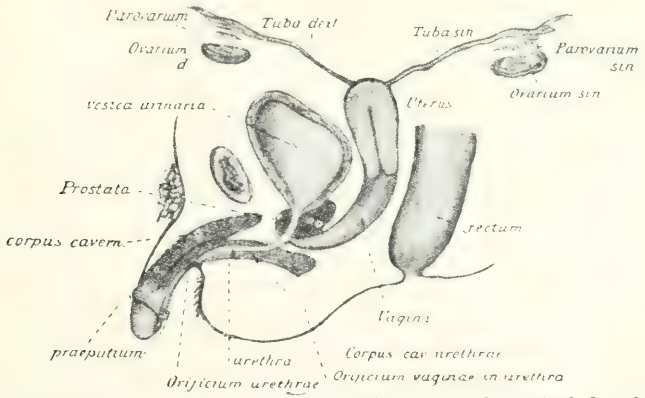


FIG. 2.—Diagrammatic sagittal mesial section of atypical female *sex-ensemble.* Note the vagina ending in the prostatic sinus; the prostate and the simulated male hypospadias. There is thus in the atypical female *sex-ensemble* no *external* vaginal aperture (Fibiger).

[*Between pp.* 328-29.

Fig. 1.—Genital organs, kidneys and suprarenal organs, from a six weeks' infant with atypical female *sex-ensemble* (female pseud-hermaphrodite). The kidneys and suprarenals are cut coronally, and each half shown. From below up; anus, labia majora, glans clitoridis, and prepuce; appearance of a male hypospadic. The fundus uteri is seen above the external genitals on the left side. The kidneys and suprarenals are above on each side. (*Photograph from glass-jar preparation by John Fraser and Carnegie Dickson.*) (¼.)

sex-ensemble; potent sex-duct elements diminished and not at the maximum of the typical male *sex-ensemble*; secondary sexual characteristics non-congruent.

Casuistic.

Cases illustrating the atypical female and male *sex-ensemble* must now be given, and the factors in the *sex-ensemble* in each summed up in the following classification.

With this classification as a catalogue, the Casuistic of atypical female and male sex must now be considered. This is enormous, but it is fortunate that it has been described, considered, sifted, and classified by Dr F. L. von Neugebauer of Warsaw in a most scientific and painstaking manner in his monograph, "Hermaphroditismus beim Menschen," a task on his part that has rendered this part of the investigation much easier and more satisfactory than it would otherwise have been. I have also drawn on many striking works and papers by Fibiger, Tuffier and Lapointe, Cullen, Goffe, C. Martin, Tandler, Grosz, Halban, Pozzi, and many others too numerous to mention.

I have, however, in illustration of my views, merely selected cases sufficient for the purpose. I have specially to thank Mr John Fraser and Dr Carnegie Dickson for permission to examine and use in illustration a specimen of atypical female sex with the usual enlarged suprarenals (Pl. II., Fig. 1).

I. Atypical Female Sex-ensemble.

This is rarer than the atypical male *sex-ensemble* in the proportion of 1 to 10, and is more difficult to diagnose. Indeed cases have been recorded where such individuals, although possessing uterus, ovaries, tubes, upper two-thirds of a vagina with an external hypospadic condition like a defective male, have worked as men and lived with women as their husbands.

The best single paper on this division is by Fibiger of Copen-

hagen, while Somerton Clark and others have recorded cases. All these have usually been cleared up, not by clinical examination but by abdominal section or post-mortem.

Neugebauer in his monograph gives statistics of such cases, and found 128 atypical female to 842 atypical male.

Fibiger gives a careful account of three atypical female *sex-ensemble* cases which he was fortunate enough to meet with in two years (1901-1903) and to examine by post-mortem. I quote nearly in full the most remarkable; the two others merely need comment.

Fibiger's Case 2 (*op. cit.*, p. 9 *et seq.*) is as follows:—

N. N., æt. 47 years, was admitted to a Copenhagen hospital, and died after being 66 days under treatment for pemphigus foliaceus and fibrinous pneumonia. The patient had enjoyed good health up till ten years before his present illness, but had not fulfilled his military service.

On *sectio* it was noted that there was a scanty full beard of thin fine hair; the hair on the pubes did not pass up towards the navel, as is usual in the male. The hair of the head was short, thin, curly, and turning grey.

The *external* genitals had a male appearance. The apparent phallus with glans and prepuce was 4 cm. long on the dorsal aspect and 3 cm. on the ventral. The prepuce was seen on the dorsal and lateral aspects of the glans, which was not completely covered by it. There was hypospadias of the second degree. The urethral cleft began at the apex of the glans and passed along the under surface to the root, where, $2\frac{1}{2}$ cm. from the apex glandis it became continuous with the urethra, and a catheter could then be passed at this point into the bladder (Pl. I., Figs. 1 and 2).

Below and beneath the urethral orifice was a hair-covered area $5\frac{1}{2}$ cm. long by $4\frac{1}{2}$ cm. broad, an arched portion with a hairless mesial groove 1 to 2 cm. broad and 6 mm. deep, ending posteriorly in a slight *raphe perinei*. The arched area corresponded to a weakly developed and small scrotum divided by the above-mentioned hairless groove.

There was no trace of a vaginal entrance, or of testes,

epididymes, and cords, either in the split scrotum or inguinal canal.

When, however, the abdomen was opened the true pelvis was found to contain apparently quite normal female pelvic organs (Pl. I., Fig. 1).

In the usual site there was a somewhat small uterus and normal broad ligaments, tubes, and ovaries; pouch of Douglas and vesico-uterine pouch normal, as well as the round ligaments. No trace of testes, cords, or epididymes. The apparent phallus had three corpora cavernosa. The corpus cavernosum urethræ surrounded the urethra. The bulb, mm. bulbocavernosi, and ligamentum suspensorium were well developed. To the bulb the membranous urethra succeeded, and then came the prostatic urethra surrounded by *a well-developed prostate, from apex to base* $2\frac{1}{2}$ *cm., transversely* 3 *cm., and in thickness at most* 1 *cm.*

The tissue was firm and white, with many small brown corpora amylacea. An evident middle lobe was not developed.

On the floor of the urethral mucous membrane were the small openings of prostatic tubules.

No traces of vasa deferentia, vesiculæ seminales, or of Cowper's glands. On the floor of the urethra between the membranous prostatic portions a 12 mm. groove (Spalte) could be seen, covered with mucous membrane and with lateral folds. Other folds gave an appearance like the male colliculus seminalis. Bladder, trigonum Lieutaudii, and ureters normal. Through this cleft (Spalte, see above) a catheter of 4 mm. diameter could be introduced. This was the vaginal entrance, passed behind the prostate, and was bounded above by the uterus (Pl. I., Fig. 2).

The entire *vagina* was $7\frac{1}{2}$ cm. long, and from its narrowed beginning (see above) became rapidly broad, so that its periphery at the prostatic level was 5 cm., and at a higher point 9 cm. The mucous membrane was in its lower part uneven, finely granular, and wrinkled; posterior fornix shallow.

The *uterus* was $5\frac{1}{2}$ cm. long and practically normal. No trace of Gartner's canals. *Tubes* normal; small Morgagni's hydatids present; epoöphoron evident, especially on the right

side. The *round* and *broad ligaments* normal, and no accessory suprarenals were present.

The *ovaries* puckered, and with stroma as in elderly women. Tunica albuginea present, with distinction between cortex and medulla. One undoubted Graafian follicle was present in the cortex, but appearances due to atretic follicles were seen. No corpora albicantia such as follow ovulation were noted, but hyaline tissue in parts may represent these (Pl. I., Fig. 1).

Both *suprarenals* were of the usual shape, but extraordinarily large, with a breadth on section of 8 cm., length 5 cm., and a thickness of 3 cm.; after hardening in formalin they weighed 29 to 30 grms. At some parts the boundary between the cortical and medullary portions could not be made out. On the external aspect of the left suprarenal a minute accessory one could be noted (Pl. III., Fig. 1).

The condition of the other organs has no bearing on the genital condition.

The *larynx* was of the female type, and with no prominent pomum Adami. Measurements are given.

The ossification type of the *thyroid cartilages* were male in type (Pl. III., Fig. 2). It may be noted here that the ossification of the thyroid cartillages is more complete in the male, and that from the larynx and thyroid cartilages Berthold diagnosed a supposed female as an atypical male. Pelvis male, except that the iliopectineal-sacroiliac measurement was female.

The *life history* of this individual was of great interest, and as follows:—

N. N. had always been considered a male, and had married a woman in his 28th year. After death it was whispered that he had been "kein richtiger Mann," and his mother had said the same to her daughter-in-law. The patient stated that he had strong sexual feeling both to his wife and other women, and to his wife three children were born. On this point the widow's statement may be quoted in her own words: "Seine Witwe teilt mit, dass er häufig Kohabitationen gefordert habe, selbst noch, als (even, still, when) er während seiner letzten Krankheit zu Hause behandelt wurde.

FIG. 1.—Right kidney and large suprarenal in Case III. (six and a half weeks' infant); both are seen in section and in natural size (Fibiger).

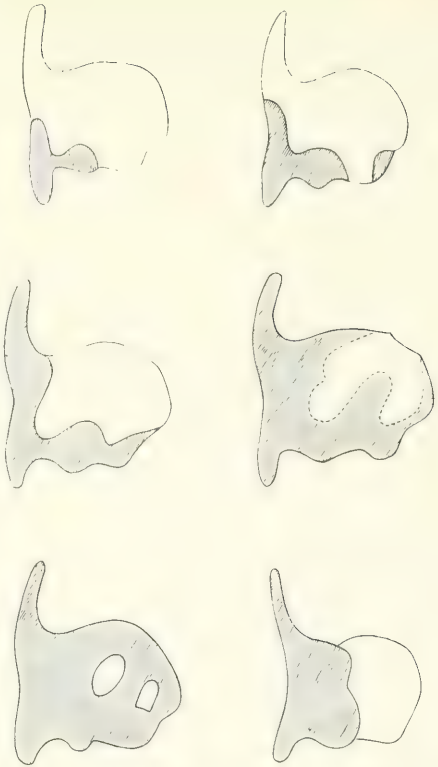


FIG. 2.—Shows stages of ossification of thyroid cartilages; lower left is male and complete; lower right, female and incomplete (Berthold, from Chievitz).

[*To face page* 332.

"Sie beschuldigt ihn sogar, in intimem Verhältnis zu anderen Frauen gestanden zu haben.

"Sie gibt aber ferner zu, dass er nicht der Vater zu zweien ihrer Kinder sei, und gesteht auch endlich ein, dass auch das dritte Kind einen andern Vater haben könne, da sie vor dieser Schwangerschaft mit einem Knechte, der auf demselben Hofe diente wie ihr Mann, in sexuellen Verkehr gestanden habe (*op. cit.*, p. 17). . . . Er trank Schnaps und rauchte gern Tabak."

There was never any menstrual flow.

Case 1 is similar, but the individual was unmarried, and died of phthisis at 58. He resembled Case 2 given above, and the suprarenals were also large. The larynx was of the male type, and the pomum Adami prominent, with the ossification of the thyroid cartilages also male.

On serial section the *ovaries* (1100 sections) showed no follicles, corpora lutea, traces of hæmorrhage, or large corpora albicantia. Epoöphoron present on both sides; no Gartner's canals. The pelvis was male in type.

Case 3 was that of an infant six and a half weeks old. The external genitals seemed male, and she was named accordingly. The right *suprarenal* measured longitudinally $4\frac{1}{2}$ cm.; the left, the same; corresponding breadths $3\frac{1}{2}$ cm. and $2\frac{1}{2}$ cm. There was a quite evident middle prostatic lobe. No testes, epididymes, or vasa deferentia present. For other features, see Table I. The *ovaries* measured as follows: Left, $17 \times 3 \times 6$ mm.; right, $15 \times 3 \times 3$ mm. Microscopically they were quite normal and full of primordial follicles. No Gartner's canals found.

Practically, then, these three cases were similar. In Case 2 one Graafian follicle was present, but in the infant, ova were abundant.

Cases 1 and 2 were at or about the menopause.

Table I.

	Body Length.	General Appearance.	Trunk.	Extremities.
Case No. 1 .	143 cm.	♂	...	Slender and full ♀
Case No. 2 .	151 cm.	♂	♂	Slender and full ♀

From a study of these cases it will be seen that there was a diminution in the potent portion of the urinogenital tract, viz. in the urinogenital sinus and urethral floor, and an increase in the non-potent, viz. in the prostate.

The secondary sexual characteristics are non-congruent, as the pelvis is male in Case 1, the larynx female in Case 2. The psycho-sexual feeling was inverted in Case 2 (see Table I.).

Table I.—*Continued.*

	Beard.	Pubes.	Pelvis.	Larynx.	Mammæ.
Case No. 1 .	♂	...	♀	♂	♂
Case No. 2 .	♂	♀	♂	♀	♂

The above gives the relations of the secondary sexual characters to a limited extent. (See Pl. III., Fig. 2, for larynx ossification.)

Anatomical Nature of Feminine Atypical Sex-ensemble.

Figures 1-4 in Fibiger show the anatomical features in such cases. The sinus portion of the vagina ends in the prostatic urethra as in the male colliculus seminalis (Pl. I., Figs. 1 and 2).

The presence of the *prostate* in atypical female sex is remarkable. In the typical female *sex-ensemble*, Skene's ducts in the urethra represent the male prostate in all probability, but in atypical female *sex-ensemble* the prostate is male in type, and is the balancing factor *re* the loss in the prostatic sinus condition, and also in the psycho-sexual attributes.

From the point of view of comparative anatomy it is to be noted that there is no prostate in monotremes and marsupials (R. Owen).

In the normal human female the prostate is represented by Skene's tubules. We may suppose that in atypical female *sex-ensemble* there is an undue distribution of a prostatic growth hormone, and they thus develop as in the normal male.

The *suprarenals* are large in all atypical cases of female *sex-ensemble*. The microscopic condition is irregular. In Cases 1 and 2 the condition was one of hyperplasia equally affecting medulla and cortex; in Case 3 the cortex was more affected than the medulla. This enlargement is not accompanied by any precocity of sexual development except in a disturbance of the hair relations. In suprarenal tumours the local sexual development is precocious, as has been shown by Glynn of Liverpool (Pl. II., Fig. 1, and Pl. III.)

II. Atypical Male Sex-ensemble Male (Pseudo-hermaphroditism).

The factors in the typical male *sex-ensemble* must first be recapitulated. These are: (1) *Potent*, descended testis and atrophied gubernaculum, with complete development of tubules, sperm-cells, vasa deferentia; prostate and Cowper's glands; phallus; normal suprarenals. (2) *Non-potent* or opposite sex-duct elements, hydatid testis, and prostatic utricle. (3) The secondary sexual characters are congruent, and comprise male type of pelvis, hair distribution, smallness of mammæ, largeness of vocal cords, and complete ossification of thyroid cartilages; psycho-sexual feeling to female.

In the normal male, therefore, the potent sex-duct elements are at a maximum of segments, the non-potent at a minimum, the secondary sexual characters congruent, and the ratio of potent to non-potent 32 : 4.

In the atypical male *sex-ensemble* we have this ratio disturbed,

owing to a greater expression of the opposite sex-duct elements or from a female type of certain structures (pelvis, larynx, etc.). Thus the testes may be undescended and like ovaries in position (Luksch's case), or descended in simulated labia majora, really split scrotum. The urinogenital sinus may be present, sometimes large, may thus be considered a vagina, and has been termed the vesicula prostatica. It is really the sinus portion of the normal vagina, the normal Müllerian portion being absent. The *phallus* may be like a hypertrophied clitoris or may be like that of a genital male, or hypospadic and simulate a clitoris. Where the sinus vaginæ is capacious and the phallus large though defective, an atypical male may act *in coitu* as male or female, and may be potent to the female. The thyroid cartilages are more fully ossified in the normal male, and thus in some atypical male *sex-ensemble* cases the supposed female, really an atypical male, has had the real sex diagnosed by laryngoscopic examination and the use of the X-rays on the thyroid cartilages. This was done in a case by Berthold, who records a case in an individual, an apparent female, brought up and dressed as such. This case may be noted here. On laryngoscopic examination by Berthold the vocal cords (Stimmlippen) were found broad and long as in big men. The epiglottis was infantile, compressed from left to right, and the pomum Adami feebly developed. The voice was manly (Pl. III., Fig. 2).

On examination the pelvic organs were as follows:—Phallus well developed, hook-shaped, and bent downwards, imperforate, hypospadias being present; there were defective labia majora and minora. When asked as to sexual feelings the patient was puzzled, and when informed as to the true sex, "machte sie ein ganz erstauntes ungläubiges Gesicht."

A further appointment was made in order to have Virchow's opinion, but this engagement was not kept for seven years, when he turned up, and then the male nature of the larynx was

fully established by examination of the thyroid cartilages (X-rays); a testis was found in a left hernial sac. Sexual inclination had declared itself, as he had "ein grosses kräftiges Mädchen zur geliebten und will sobald wie möglich heirathen." The whole diagnosis was fully confirmed by the demonstration of well-developed spermatozoa in an ejaculation.

Exact anatomical details of the condition are rare, but in some cases these have been recorded, and will now be considered along with some others where operative work was done or where the clinical examination was thorough. The illustrative cases will be taken relative to the exaggerated opposite sex-duct elements and from below up. The cases of Neugebauer, and especially one by Tuffier and Lapointe, will be studied to illustrate the inverted psycho-sexual conditions sometimes found.

Case I.—Christopher Martin of Birmingham has recorded a case of atypical male *sex-ensemble* where the external genitals and vaginal entrance were those of a female (Pl. IV., Fig. 1), and the testes were in the inguinal canal. The clinical history is as follows:—

Mrs P. (?), æt. 44, married twenty years, consulted Martin for inguinal tumours, which on operation proved to be testes, the interstitial cells being markedly developed but the tubuli filled with a delicate connective tissue. In some of the tubuli spermogenic cells were found. The epididymes were more or less normal. The vas deferens was traced down behind the bladder at the operation. The voice was feminine, and the patient shy and timid. The vaginal *cul-de-sac* was 1½ in. long, and hymeneal remains were noted.[1]

Coitus had been satisfactory on both sides. There was no representatives of uterus or tubes. Here the opposite sex-duct elements unduly developed were the sinus portion of the vagina, the hymen, and the mammæ. This gain was compensated by the split scrotum and the imperfect condition

[1] Carunculæ myrtiformes are found after labour.

of the phallus and urethral floor. The psycho-sexual condition was inverted. Probably the larynx was female, but the thyroid cartilages were not X-rayed; they were evidently of opposite sex type.

In this case, therefore, the ratio disturbance affected mainly the lower ends of the duct tract, viz., urinogenital sinus, hymen; the mammæ and psycho-sexual condition were also of opposite sex type. The hymen was present, as it is not a Müllerian product but an organ of the urinogenital sinus and Wolffian ducts. Although the testes were descended, the sperm-cells were defective, as I found in a case of atypical *sex-ensemble* in the pig.

Case II.—Neugebauer records a case (*Hermaphroditismus*, p. 885) where an atypical male discharged the duties of a wife to a widower to whom she was married, where the urinogenital sinus was somewhat developed and acted as an imperfect vagina, but the spouse (?) was sexually apathetic and suffered pain on coitus. The husband was satisfied with his mate, "mit der körperlich sehr kräftigen Frau die ihm im Felde und Walde und im Hause männlich schwere Arbeit verrichtet." The details are as follows:—A. G., 172 cm. tall (5 ft. 8 in.), bones and muscles male, body and genitals hairy, hair of head short, appearance of face male, larynx and voice male type, shoulder-breadth greater than that of pelvis. Hypospadias and split scrotum present. Phallus, 6 cm. long dorsally, 5 cm. ventrally, and 5½ cm. in circumference, that of a twelve-year-old boy, with prepuce superior and lateral only.

A vestibule was present measuring 25 mm. vertically; labia minora and majora small. A sex gland (testis) was present in each labium. Below, the urethra, vaginal entrance, and torn hymen could be made out. Into the vaginal sinus the finger passed 6 cm. No uterus and no prostate present, but possibly a rudimentary uterus and tubes. The spermatic cord could be rolled under the finger and against the horizontal ramus of the tubes.

The patient took her condition philosophically, and remarked, "Wenn sie Gott geschaffen habe, so werde kein

Artz daran etwas ändern." His reluctance to clinical display was overcome by the gift of a few roubles.

CASE III.—This case, by Leopold of Dresden, is interesting as to the existence of the urinogenital sinus. The patient, N., 46 years of age, brought up and dressed as a girl, and unmarried, wished her sex determined, as for three months she had had illicit intercourse and conducted herself as a man sexually.

N. was male in type, 174 cm. tall (5 ft. 9 in.), with much rough hair on face, rough deep voice, and genitals hair clad, with an upper convex border at the pubes. The phallus was 6 cm. long, of the usual male calibre, imperforate, and with a circular prepuce not covering the glans.

Instead of a urethra there was an open groove from the glans apex to the lowest part of the phallus root, where it widened out and opened. A catheter passed *viâ* this opening entered the bladder. Below this was an opening into which a catheter passed 4 to 5 cm. This canal ended blindly. There was no scrotum, but labia instead, and in them descended testes. No labia minora. There were erections now, but even *in coitu* with a well-developed girl there was no effective result, and the same was the outcome of coitus with a lad. In his seventeenth year apparent menstruation came on every four weeks, lasting three to four days. Varices of the limbs were present since his twentieth year, and then, according to his statement, "ein Bursche mit ihr den Beischlaf häufig angeübt habe welcher aber später und überhaupt bis jetzt nie wieder von einem Manne mit ihr vollzogen worden ist."

The existence of the urinogenital sinus, a canal common to both sexes, allowed of a certain amount of male intercourse, and the incomplete phallus of the same, with a female, Carlyle's "problematic Chevalier d'Eon, now in petticoats, now in breeches." Leopold considered what I have called urinogenital sinus to be partly Müllerian, terming it vesicula prostatic. The prostatic utricle in the normal male represents the hymen and sinus too, and has the vasa deferentia opening in it.

This case may be regarded, then, as one of atypical male *sex-ensemble*, with the urinogenital sinus simulating a vagina, and the hypospadias the feminine external parts (text figure 1).

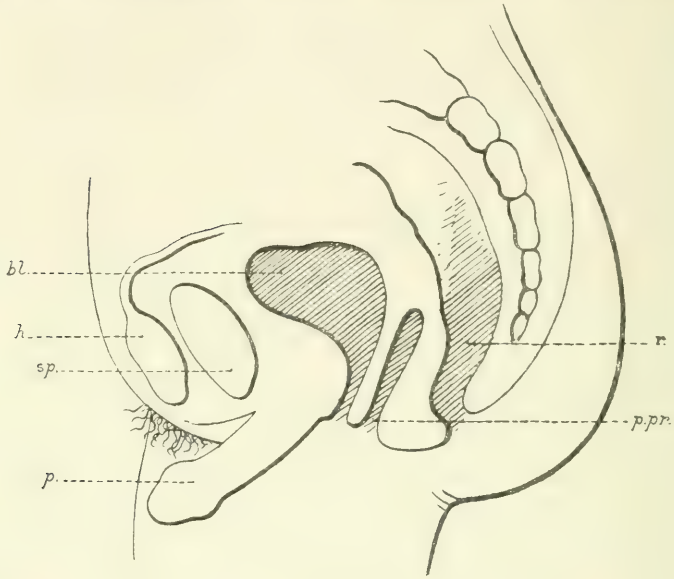


FIG. 1.—Leopold's Atypical Male *Sex-ensemble*.
bl, Bladder; *h*, testis; *sp*, symphysis; *p*, phallus; *p.pr*, vericula prostatica, urinogenital sinus; *r*, rectum.

CASE IV.—This case, by Luksch, is of great value, as a post-mortem was obtained and a drawing of the internal organs is given. There was no hypospadias.

The patient was a plasterer's labourer, 45 years old, who died in a lunatic asylum, where he had been placed owing to his suffering from persecution mania. The right half of the scrotum was empty, but in the left there was a hernia and also an apparent testis the size of a bean, but this was not definite, and as we shall see, not a testis. He ultimately died from general tuberculosis.

The section gave the following:—Body, 5 ft. 9 in. in height, habitus male, bony skeleton tolerably well developed, muscles feeble, hair of head thick, beard and hair on lips and cheeks.

The pubes and phallus were normally male, scrotum empty, but a hernia, as already stated, on the left side.

The prostate and caput Gallinaginis were of the usual size, and the same applies to the urethra, corpora cavernosa phalli, and the corpus cavernosum. Cowper's glands were small.

Behind the bladder, and attached to it by loose connective tissue, was an apparent uterus (erhob sich ein uterus ähnliches Gebilde), its fundus projecting above that of the former by 1 cm., and 10 cm. above the upper edge of the prostate. From the fundus there were two short horns to the left and right; from the horns, two Fallopian tubes, the left 11 cm., the right 7·5 cm. The uterus and tubes were defective, but details are unnecessary. It may be noted, however, that the vagina opened in the sinus pocularis as in the cases of atypical female sex (Pl. I., Fig. 2). On the left side, part of the tube, the round ligament, and the left horn were in the scrotal sac, while in the right one the round ligament and the right cornu lay.

The *sex-glands* were testes, but the tubuli seminiferi were merely thin threads. No ovaries were present. A vas deferens was present on each side, which ran on each lateral aspect of the uterus and ended in the cellular tissue of the mesometrium. On the left side it ran into the hernial sac; on the right to the neck of the sac. Above, each ended in the connective tissue of the tubes. They then pass in the line of the tubes to the testes. Vesiculæ seminales were present.

From his excellent drawing we can see that the *potent organs*, testes, vas deferens (?), vesiculæ seminales, prostate, Cowper's glands, phallus, the gubernaculum, were not functional, as the testes were undescended, and the vasa deferentia, too, were defective. The *opposite sex-duct elements* were greater than in the typical male *sex-ensemble*, as instead of the prostatic utricle and the hydatid testis there were present uterus, cornua, tubes, and vagina, although in a somewhat aplasic and malformed condition. The increase in the non-potent organs was compensated by the absence of the gubernacula, and by defective segments of the vasa deferentia. There was thus here a disturbance of the maximum-minimum

ratio, the latter being increased at the expense of the former. No psycho-sexual facts were obtained, but it must be noted that the man was insane and tubercular.

While there is this disturbed ratio it must be noted that the balancing factors are irregular in each case. The testes may be descended (often late) or undescended; the scrotum may be male, or split as in the female; hypospadias may or may not be present. The psycho-sexual feeling may be inverted, apparently awanting, or the individual may attempt sexual relations with both sexes.

These, however, are really the various results one may get in a probability distribution of the various factors making up the sex duct and psycho-sexual conditions in male and female.

Case V.—This is recorded by Tuffier and Lapointe, and is, in its psycho-sexual aspects, a most valuable one.

Mlle. S., aged 20, consulted at the Beaujon Hospital as to the removal of a swelling in what appeared to be the left labium majus. The motive of this consultation was in view of her approaching marriage.

The clinical history was that at sixteen this swelling appeared in the left groin and gradually descended to its present position. No pain accompanied this, and the removal of the tumour was requested on æsthetic grounds. On the right the corresponding swelling was less prominent. The swellings felt like testes with epididymes, and there was no trace of hernia. No cremasteric reflex was present. The so-called Mlle. S. was thus a man.

To make the diagnosis certain, the left demi-scrotum was incised, the testis and epididymis exposed, and a piece of the testis removed for microscopical examination. Plate IV., Fig. 2, shows the actual external condition. A urethral orifice was present, but no vaginal one, and no uterus and appendages were found, but an apparent prostate could be felt.

The testis was found to be defective in structure, especially in the sperm-cells, but the interstitial cells were numerous.

The important point now comes up as to the *sex-ensemble*, and

Fig. 1.—External genitals from case of atypical male *sex-ensemble* recorded by Martin of Birmingham. Anus, fourchette, hymen, labia majora and minora, vestibule are seen. The testes were in the labia majora, and the individual had acted as wife.

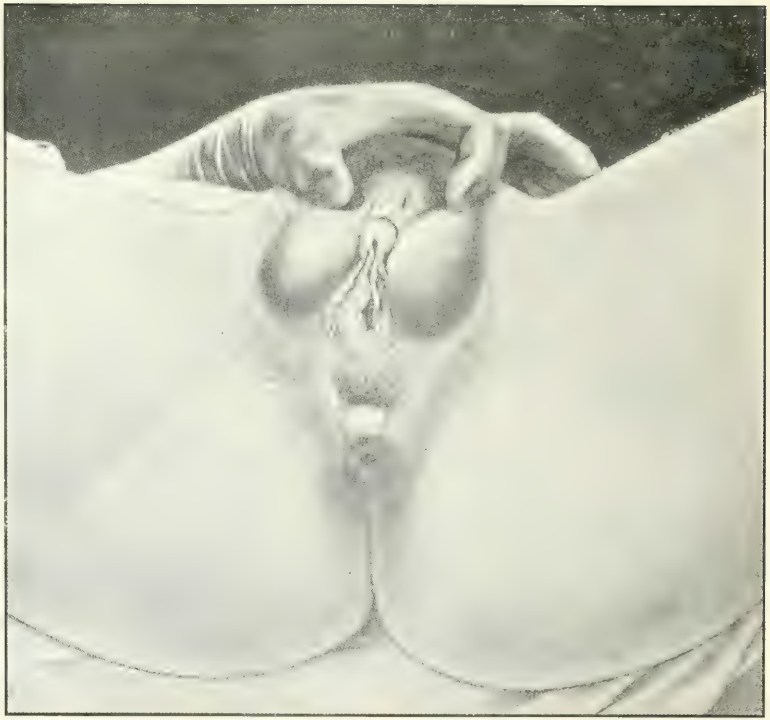


Fig. 2.—External genitals in Tuffier and Lapointe's case of atypical male *sex-ensemble*. There is no external aperture but the urethral one, and the testes are in the split scrotum.

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it is in it that we see the remarkable nature of the *sex-ensemble* variation and also the importance of studying such, not from the point of sex merely, but from the broader one of *sex-ensemble*. The secondary sexual parts are mainly feminine, and the psycho-sexual feeling for the male; the patient is frankly an invert in this respect.

When dressed Mlle. S. seemed to be an undoubted female of graceful and attractive figure. The timbre of the voice was feminine, and the appearance of the neck, hair, shoulders, and trunk were the same. The hands, lower limbs, feet, and ankles were large and male; the hips prominent and female.

The mentality was feminine, with an accurate and lively sense of all that pertains to feminine toilette, quite in contrast to one recorded by Neugebauer, where the attempted feminine attire was crude and glaringly out of taste.

The answer of Tuffier's patient to the question if he was capable of understanding the necessary transformation was: "Pourquoi ne pass m'enlever cette horrible grosseur de l'aine gauche qui me gênerait si je voulais poser chez les peintres? Puisque vous ne pouvez rien pour moi, j'irai trouve le célèbre chirurgien D——, qui saura bien faire de moi une femme! Si j'étais comme les autres femmes, je me serais mariée et je n'aurais pas plus besoin de travaillier pour vivre." As to sexual feeling, the statement was: "Sans réticence, il a des désirs génésiques qu'il satisfait solitairement ou avec des hommes; jamais avec des femmes. . . . Les excitations génitales amènent l'érection de son pseudo-clitoris; elle sont suivies de sensations voluptueuses et d'éjaculations qu'il ne nous a pas été possible d'examiner au microscope. Dans ses relations génitales L. S. est franchment homo-sexual et ne cache pas son goût pour les hommes . . . a dixhuit ans, première tentative de coït comme femme, dont l'insuccès le surprit à peine 'parce que, dit il, c'était avec un homme déjà mûr et je savais que le virginité de certaines femmes ne cède pas au premier contact.' . . . Il affirme qu'il ne s'est jamais livré à la pédérastie passive et il n'en présente aucun stigmate."

We have here, then, a remarkable variation in the atypical male *sex-ensemble*. A male by sex gland, somatically in most

respects feminine—pelvis, hair distribution, mammæ, trunk; in other respects male—hands, lower limbs; above all a female psycho-sexually, and thus an invert, we see here a variation result in mankind, where male and female sexual traits are so distributed that we cannot but pity this sport of the gods and fate. Truly, the probability mechanism of evolution has made mock of this man and evolved a cruel mixture of the sexually dissatisfied and unsatisfiable.

The question of DIAGNOSIS must now be taken up.

In *atypical female sex-ensemble* the diagnosis is difficult if not impossible in the *infant*. In Fibiger's case (2, p. 330) the diagnosis was hypospadic male, and in Carnegie Dickson and Fraser's case it was the same; yet both were females. In the *adult* the presence of the prostate and of the hypospadias misleads, and in Fibiger's Case 2 the real diagnosis was impossible. Then, again, the secondary phenomena are non-congruent, and this further complicates diagnosis. Thus the thyroid cartilage ossification may be male or female, and sexual inversion may be present.

One point must be noted, however, that bimanual examination may reveal the presence of the uterus and sex gland. In Luksch's case (p. 340) with an atypical male *sex-ensemble*, the opposite sex-duct elements were expressed as uterus and tubes, and this must be noted. The real diagnosis is thus only settled by the nature of the sex gland, and therefore an incision into the demi-scrotum and removal of a piece of the sex gland for microscopical examination may be necessary. Luksch's case would have been very puzzling without a microscopical investigation, as the sex glands were in the position of ovaries, undescended.

In *atypical male sex-ensemble* the diagnosis is easier. At first sight the *infant* may appear female, especially if there is an aperture between anus and urethral orifice simulating a vaginal entrance. The split scrotum will support the diagnosis

of female, but if the testes are in the demi-scrotum they should be carefully palpated for the cord which can be rolled against the pubic ramus.

In the *adult* the diagnosis will be easier, especially if the testes are descended, and then the cord may be felt. When the lower part of the urinogenital sinus is present it may mislead, but it is a *cul-de-sac*, and the other features of the *sex-ensemble* should be very carefully and thoroughly examined—hairiness of body, nature of voice, ossification of thyroid cartilage, nature of mammæ, of bones and muscles, and of pelvis. The psycho-sexual feeling may give aid, but it is to be remembered that it may be inverted, indifferent, or hetero-sexual (see Tuffier's case and those of Neugebauer, pp. 338 and 342).

In infants the diagnosis may be so difficult as to prevent a correct diagnosis being made. *Such cases should be brought up as boys.* The chances are ten to one that the sex gland is male, and if brought up as females they may at puberty cause scandal with the opposite sex. That such should be brought up as males was Lawson Tait's advice.

Some *miscellaneous points* may now be noted.

In *atypical female sex-ensemble* cases menstruation does not occur. In male atypical cases there is sometimes a pseudo-menstruation, as in a case recorded by Neugebauer and others (*op. cit.*, p. 236, *Hermaphroditismus*).

Homo-sexuality.—This interesting, if somewhat repulsive subject is illustrated in what may be called its physiological and mental aspect by some of the cases.

Fibiger's Case 2 showed the rarer form of inversion in the female atypical *sex-ensemble*, and Neugebauer's case (p. 338) exhibited the same phase of sexuality. In both of these it was unconscious, *i.e.*, they obeyed the exaggerated opposite sex psycho-sexual element distributed to them. Tuffier's and Lapointe's case shows homo-sexuality in a most pronounced

form, and its cause was the evident opposite sex psycho-sexuality present. He was thus a victim of his organisation, and not a depraved personality, as may sometimes be the case. This should be kept in mind in judging unfortunates with homo-sexuality; they are cases for mental treatment by the judicious specialist, and not in the ordinary "Depart from us, ye cursed" way usually meted out to them. The whole question of the treatment, moral and punitive, of homo-sexuality is, however, most complex and unsatisfactory.

Treatment in Atypical Sex-ensemble.

On the whole there is no treatment. In cases of mistaken sex, usually in atypical male cases, the sex may be declared, and the necessary legal steps taken. In Croom's case, where two atypical brothers had been brought up as girls, this was done, and they went abroad. It is questionable if cases like C. Martin's and Neugebauer's (pp. 337 and 338) should be interfered with, especially if up in years, and there is mutual agreement. It is not advisable to remove the sex glands, as they are important internal secreting organs, nor to amputate hypertrophied phalli.

In J. R. Goffe's case the patient was an apparent female with male hair arrangements and phallus much enlarged for a female; hypospadic, and with an apparent rudimentary vagina. Goffe removed what seemed to be an enlarged clitoris, dilated the rudimentary vagina, and ingeniously used the mucous covering of the clitoris to re-line the sinus. The result was immediately good, but one has serious doubts as to whether the patient was not male after all. Perhaps the real nature of the case may be cleared up by a late descent of the sex glands.

In atypical female *sex-ensemble* nothing can be done. The vagina ends in the prostatic urethra, and is thus inaccessible.

The following is a summary of the view taken as to atypical *sex-ensemble*:—

1. The atypical or pseudo-hermaphroditic *sex-ensemble* case is either male or female, and in this is judged by the nature of the sex gland.

2. The potent, non-potent, and secondary sexual characters are not in the maximum-minimum ratio with congruence of the secondary sexual characters.

3. The non-potent in atypical *sex-ensemble* are thus increased, and the congruence of the secondary sexual character is disturbed; this is characteristic of a probability result.

4. It is to be specially noted that in the atypical female cases a prostate with lateral lobes only (Fibiger's Cases 1 and 2), or with all the lobes (Fibiger's Case 3, an infant of six weeks), may be present.

5. In the atypical female cases the suprarenals are enlarged in all the accurately recorded cases (Fibiger's and Fraser and Dickson's), but the bearing of this in such cases is not accurately known.

6. In male atypical cases part of the lower urinogenital sinus may be present, and may thus simulate an imperfect vagina; sometimes a hymen is present, and in Christopher Martin's case the external genitals and vaginal entrance resembled those of a female in every detail.

7. Certain atypical male and female *sex-ensemble* cases may be "inverted" both in sexual feeling and in mentality.

8. In atypical male *sex-ensemble* cases the testes may be pelvic, in the groin, or completely descended into the two halves of the scrotum (Tuffier and Lapointe, Neugebauer and C. Martin).

9. In certain atypical male cases the sexual instinct may be doubly exercised.

10. In essence, such cases have this sequence developmentally — (*a*) Loss of parental determinants of the *sex-ensemble* at maturation; (*b*) the subsequent formation of

sex-ensemble molecules with this loss when the determinants are distributed in the p. g. c. mass and p. s. c. mass; (*c*) in such cases the sex gland will have normal *sex-ensemble* molecules, and others unduly reduced. The latter will give rise to atypical male or female *sex-ensemble* in progeny; (*d*) this can be distributed again by such progeny to their offspring. (See previous paper, *Edin. Med. Journ.*, July and August, 1914, p. 106.)

11. In diagnosis the whole *sex-ensemble* must be taken into account; above all, that of the sex gland when accessible. No diagnosis can be based on one organ of the *sex-ensemble*, *e.g.*, on the presence of a prostate, hymen, apparent vagina, condition of larynx, or of the pyscho-sexual feelings; the last may be inverted (see cases *passim*). The treatment is, as a rule, negative.

12. *An apparent vagina with labia means male sex-ensemble; in the atypical female sex-ensemble there is no external vaginal entrance, as it ends in the prostatic sinus.*

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The Vice-President (*Dr Keppie Paterson*) thanked Dr Hart for his paper, and said that his clear critical faculty had done something to throw light on a rather dark subject. Dr Paterson referred to a case of his own where he had been placed in a practical difficulty about the sex, where the patient brought had male mammæ, considerable growth of hair on cheeks and chin, also on the trunk; broad hands and a rather deep voice. There was to all appearance regular female organs of generation with a very thick projecting clitoris, a complete hymen, and a complete large vagina, and evidently a very small rudimentary uterus at the upper end of the vagina. There was no evidence of anything in the labia. Psychically there was no tendency towards the male sex, but the patient was fond of young children. The patient had been educated as a girl, and he considered it right that the case should be still considered a girl.

Dr Ballantyne said he had had the opportunity of reading Dr Hart's paper, and would in the first place back Dr Hart with regard to terminology, where he had made a distinct advance. But there was one matter in this connection on which Dr Hart had not touched: it was that case in which a supposed true hermaphrodite had been described in the human subject with an organ to which the term ovo-testis had been given: the one part of the gland being like an ovary, and one part like a testis. It had not been quite proved that it was truly both

ovary and testis, and he thought the probability was that it was an ovary, and that the part that was considered to be like a testis was really a malformed ovary. A good many of Dr Hart's points would depend on whether they could exclude these cases of ovary-testis. He himself personally agreed with Dr Hart that both glands were not found in the same subject. If they could come to this conclusion, then they could sweep away the cases of true hermaphroditism, and if there was no true hermaphroditism, there was no good speaking of pseudo-hermaphroditism.

His real point was as to the expression "*sex-ensemble.*" He had been trying to get an English word, such as "sex totality," but was not satisfied with it. He considered Dr Hart's classification was very simple—(*a*) atypical male *sex-ensemble*, testis present; (*b*) atypical female *sex-ensemble*, ovary present—corresponding to the pseudo-hermaphroditism masculinus, and pseudo-hermaphroditism femininus. It was a great advance to show clearly that beyond the fact of ovaries and testes there were no characters that could certainly be said to belong to male or female.

The question of the enlargement of the suprarenal glands was a difficulty. Recent investigation seemed to show that other parts of the body besides those commonly associated with sex seemed to have to do with sex. He wondered whether in these cases with enlargement of these suprarenal glands there was a return to fœtal conditions (where these glands were larger proportionately). The pineal gland also was supposed to have something to do with sex development. Dr Blair Bell said a woman was not a woman because of her ovaries, and a man was not a man because of his testes; but that a woman was a woman because of her internal secretions, and a man was a man because of his internal secretions. If this were so, it would be still more difficult to say the sex of an individual.

One cannot tell from the duct elements.

There is a question of medico-legal importance arising out of such cases. With regard to diagnosis, he supposed Dr Hart in speaking of the bimanual examination referred to the recto-abdominal. That the prostate gland was not decisive of sex was an important point.

There were some practical difficulties in these cases; what was to be done for these creatures? It was an atrocity to think of dilating up the urinogenital sinus, and carrying out such an operation as had been done in America. He quite agreed with Dr Haultain that such cases should be let alone. But there were some cases where re-declaration of sex (such as in Sir Halliday Croom's case) might be quite a good thing.

All would agree that there should be a careful examination in the new-born infant.

There was one other point in the diagnosis that was sometimes found of use: the feeling of the cord running up in the inner aspect of the pubic ramus from the body of the testis.

The explanation of all these cases by Mendelism, as suggested by Dr Hart, had not been mentioned in the paper to-night. It was the only part of all Dr Hart's full paper with which he did not agree.

Dr Fordyce had listened with great pleasure to the paper. He was quite incompetent to criticise it. He would like to ask in what proportion of these cases of atypical male *sex-ensemble* were the testicle found in the labia. He referred to a case he had recently seen in London, where there had resulted hæmorrhage from coitus, which had been stopped by plugging, and when the plug was removed it was discovered that there was no cervix, and nothing found in the roof of the vagina. The abdomen had been opened, and testis had been found in the abdomen.

He did not altogether agree with Dr Hart that the term pseudo-hermaphroditism should be objected to.

Dr Young mentioned a case he had seen when he had been Resident in the gynæcological ward, of a patient who had been married to a man for two or three months, dressed as a girl, with long hair, and looking like a girl. But there were striking characteristics of a male—broad shoulders, no mammæ, development of hair on the pubes like a male; and on examining the external genitals, there was a split scrotum with a testis on each side, and a penis, and the urogenital sinus was patent. There was an interesting after-history. The patient agreed to have her hair removed, and left hospital wearing male clothes. Later the person married a girl!

Another point he wanted to ask about, though it was hardly relevant, was as to the origin of the mammæ in the male. In the female he could understand it, but not in the male.

Dr Haig Ferguson agreed cordially with Dr Ballantyne about Dr Hart's paper. Sir J. Y. Simpson's paper, which had been the best, was now superseded chiefly by Dr Hart's work. He wished to ask one question with regard to the bimanual examination, or rather to point out that such an examination could not be conclusive, for, as pointed out in one of the cases to which Dr Hart referred, a uterus and ovaries would have been felt in the examination, and the case would be assumed to be a female, but post-mortem testes were found, and therefore the patient was a male.

Dr Hart said such an examination would only be conclusive in a case of atypical female *sex-ensemble.*

Dr Berry Hart, in reply, said he was much obliged to them for listening so patiently. The difficulty he had had was to

dispense with the material which he might have brought up, and, therefore, in shortening his papers he might have left a wrong impression in some things. As to the ovary-testis, there were tubuli seminiferi at one part, at another part there was a circular cell with a low columnar epithelium, and a cell inside it—which probably was a seminiferous tubule undeveloped, with a sperm cell inside. On the other hand, in the female, one did not find a cell with a low columnar epithelium. At some time theoretically there was no reason why there might not have been an ovary-testis. In fishes there was often an ovary-testis, but in mammals there was a complete elimination.

Dr Nagel had examined Dr Blacker's specimen, and said it was a defective testis.

As to the term "*sex-ensemble*," he quite saw Dr Ballantyne's point. By the term he meant something beyond the sex glands, and the term "*ensemble*" was used in much the same sense as he wished to use it here.

As to the enlargement of the suprarenal glands, when this enlargement was found with tumour growth in them, there was precocious development of sex. But when there was enlargement without tumour growth, there was no precocious development of organs. The internal secreting glands were of the highest importance in sex, and had been too much neglected. The suprarenals played some part undoubtedly, and he included them as part of the *sex-ensemble*. As Dr Ballantyne said, there was a part in the fœtal suprarenal which disappeared later, for which the reason was not known.

He was obliged to Dr Fordyce for his remarks. There was no harm in the term pseudo-hermaphroditism, but if there was no true hermaphroditism, he thought the former word should not be used. And further, in pseudo-hermaphroditism the duct elements were taken as the important elements in diagnosis instead of the sex glands, and he considered they were better without the term.

Dr Young had asked about the origin of the male mammæ.

In the distribution of the *sex-ensemble* he thought that perhaps there were hormones which stimulated the mammæ, but he had nothing but speculative thoughts on the subject. Every man and every woman was a mere variety derived from the species homo, but there were certain changes arising later which were complementary to one another.

He agreed with Dr Haig Ferguson with regard to the bimanual examination, and said Dr Ferguson had put his finger on a weak spot in diagnosis.

The whole question was surrounded by some difficulty. The clinical difficulties were great, and the responsibility in diagnosis considerable.

APPENDIX.

ON THE NEED FOR EUGENIC REFORM.

By Major Leonard Darwin, President of the Eugenics Education Society.

(Conjoint meeting with the Medico-Chirurgical Society to inaugurate the new meeting-place, Wednesday, 29th October 1913.)

Linnæus, writing in the middle of the eighteenth century, declared that species are now as numerous as when Providence first created them in the beginning, and that, in accordance with the laws of generation then laid down, the beings originally created produced only forms similar to themselves, with the result that the species now with us are no more numerous than they were at first. In writing thus this great naturalist was only expressing the belief in the immutability of the different forms of life almost universally held in all civilised countries until the middle of the last century. It is true that the suggestion that the world as we see it now is the outcome of a long series of continuous changes, generally similar in kind to those actually taking place around us to-day, had been made by several philosophers in ancient days; whilst from time to time some more advanced thinker would express the belief that every animal, including man, is the lineal descendant of some more primitive type of living creature. But those who held such views were always so few in number that the general acceptance of their beliefs—the practically universal acknowledgment amongst thinking men during the last fifty

or sixty years of the truth of the principle of evolution—can only be described as a complete revolution in thought. But must not the adoption of the belief that by evolution alone can we account for the visible facts of life tend to produce material alterations in the way in which we regard many practical questions? Is not every revolution in thought inevitably accompanied by more or less considerable changes in policy and social customs? The truth is that it is quite impossible for us to go on acting as if we held precisely the same beliefs as our ancestors; and the aim of the eugenist is both to guide and to hasten the reforms in social customs and in legislation which must in any case result from the acceptance of the doctrine of evolution.

But are any new methods of reform necessary besides those which we are now so earnestly striving to introduce, it may be asked? Anyone who studies our Poor Law statistics, or who looks at our ever-growing lunatic asylums, homes for imbeciles, and hospitals, or who considers our criminal records, or who merely takes a single walk in our city slums—anyone who ponders over the subjects thus brought to his mind must at all events feel profoundly dissatisfied with the existing social condition of his country. Splendid efforts are no doubt now being made on all sides; but, when we remember how long this noble strife has lasted, we are absolutely driven to inquire whether, in addition to all the good now being done, some other and hitherto untried methods are not also essential to secure the progress of the nation, or indeed to safeguard it from decadence. If, as we hold, it will be necessary to continue in the future as in the past to try to alleviate or mitigate the sufferings of our fellow-creatures by the application of immediate remedies, it will be absolutely incumbent on us henceforth also to recognise the necessity of searching for the ultimate sources of all these evils in the light of our ever-advancing knowledge of the laws of nature. In the past the efforts of the social

reformer have been almost entirely directed to conferring immediate practical benefits on man by altering his mental and physical surroundings. Without relaxing these efforts, we must also in the future endeavour to ensure that our descendants shall actually enter the world with a better natural equipment than that which we possess wherewith to fight the battle of life. As long as complete ignorance prevailed as to the way in which the generations of man succeeded each other, it was inevitable that all philanthropic efforts to help the human being should have begun at his birth. But now that this ignorance is being rapidly dissipated we are bound to see if something cannot be accomplished before his birth; and this is the aim of the eugenist. We hold that the world is so constituted that all the good and useful inborn qualities of man, as well as all his evil tendencies, have been evolved in accordance with what are called the laws of nature; and our hope and belief is that by studying those laws, and by utilising the processes thus discovered by which man has been evolved from some lower type of life, we shall now be able to take steps tending to ensure that the man of the future shall be a far nobler being than the man of to-day.

We eugenists are no doubt not yet in a position to formulate a policy with great precision; and I for one hope that we shall not attempt to do so for a long time to come; because scientific dogmas are apt to be the death of scientific progress. Moreover, when it is remembered that Sir Francis Galton, the founder of our science, wrote his first paper on this subject in the year 1865, and that we have not therefore yet celebrated the jubilee of this movement, no one ought to be surprised that more has not been done and that much remains still unknown. This fog of doubt, which is largely due to our ignorance of the methods adopted by nature in promoting racial progress, is, however, certainly slowly clearing away; and even now

through it we believe that we see quite clearly that the selection of the ancestry of the coming generations—the selection of the stock from which the race of the future is to spring—has been at all events one of Nature's main methods of ensuring racial advancement. But if Nature has always as it were paid the closest attention to the factor of heredity in dealing with all animals and plants, and if we wish to see a continuance of the progress thus made in the past, the men of the future becoming healthier, happier, and nobler than the men of to-day because of their superior inborn qualities, surely if this be our aim we clearly see that the duty of studying the question of human heredity is imperatively imposed upon us. In short, whilst we hold that the social reformer should receive every encouragement in his efforts towards the betterment of human surroundings, we eugenists are convinced that it is essential at the same time to adopt certain reforms based on the factor of heredity. Between these two movements—the environmental and the heredity—there need, we think, be no hostility whatever; for both are absolutely necessary to ensure human progress in all directions. The breeders of racehorses do not waste their time in quarrelling as to which is the more important for their horses, good training or good pedigrees; and the breeders of mankind may in large measure leave it an open question which is the more important, environment or heredity. And this is a point which needs emphasising, because misconceptions as to the opinions we hold on these questions have given rise to ill-founded prejudices against us.

The confession which I have just made that the eugenic programme is somewhat indefinite must, however, not be held to imply that there is any lack of work for the eugenist now to do. To convince our fellow-countrymen that human heredity is a subject imperatively demanding our attention; to advocate those moral and legislative reforms which even now we see to be advisable; and to continue those scientific investigations

which are necessary to enable us to advance with greater confidence in the future—here in all probability is work enough for all our lifetimes. And firmly believing as I do that all who are willing can play some part in thus helping forward this great social movement, I sincerely trust that a eugenic society may be founded in the capital of Scotland, so long the seat both of morality and of science.

My object to-night will merely be to briefly allude to some of the unsolved problems of eugenics, and to indicate some of the ways in which eugenic societies can aid both in their solution and also—and this is perhaps their more important function—in influencing public opinion in the desired directions, in the hope of thus promoting the necessary moral and legislative reforms. But before doing so I should like to suggest to those joining this society that it is well to take some little pains to grasp what is really included within the scope of the science it is especially intended to promote. All who sympathise with this movement will probably have read the well-known definition of eugenics finally adopted by Galton, namely, that it includes "the study of the agencies under social control that may improve or impair the racial qualities of future generations, either physically or mentally." Now that definition might at first sight appear, to those who have grasped its full meaning, to be all that is needed both to avoid misconceptions and to attract the attention of the public to this subject. Experience, however, proves that a definition standing alone is almost useless; because, amongst other reasons, the meaning it conveys depends in great measure on the mental atmosphere, so to speak, of the inquirer. This definition has been often wholly misunderstood, whilst perhaps still more frequently it has resulted in the mind being focussed on one only of the many branches of this subject. For example, the words "racial qualities" will forcibly recall to the minds of many social reformers what have been described as "racial poisons," including alcohol, lead, etc., and may thus

lead to the belief that eugenics is mainly concerned with the cure of the undoubted evils arising from such causes. Without going deeply into these vexed questions, a word or two as to how far this view is justifiable may be advantageous.

Though the direct evil effects of alcohol, on those who take it in excess, at all events, as well as the moral and material deterioration of their families, are quite unquestionable, yet the eugenist, looking to the nation in the future, must also inquire whether moderate or hard drinking does affect the descendants of the drinker or the drunkard as regards their innate qualities. On this point we are still in doubt, although many confident assertions have been made. There is, it is true, some evidence to show that the sons and daughters of a drunkard are thus damaged as regards their inborn qualities; but, if we look to the actual biological effects on the third and subsequent generations, we are, in my opinion, in complete ignorance. Moreover, as regards those dread contagious diseases which produce such certain and terrible effects on the children of the diseased, here again the biological consequences to future generations are quite problematical. It seems, then, that the damage done both to the current generation and to their unborn children alone affords ample justification for a campaign against drink and disease, a campaign in which the eugenist should be all the more willing to join because it may yet be proved that these "racial poisons" do injuriously affect posterity for an indefinite number of generations, in which case they would be shown to be eugenic factors of prime importance. We must not, however, neglect the other possible alternative, and we ought to admit that, however vast would be the immediate benefits resulting from the abolition or destruction of racial poisons, yet it may well be that this would not be a step tending to produce a progressive advance in the inborn characters of mankind, which is the true aim of the eugenist. If this be so, surely it follows that, without relaxing our efforts

in this noble social campaign, we yet should not remain satisfied until we have ascertained whether some steps cannot be taken which would unquestionably produce that most desirable result.

All around us we see efforts being made to improve the environment of the people, efforts with which I have already expressed my sympathy in the most emphatic manner. But with reference to reforms of this type, if we look the facts of life in the face, we must acknowledge that as regards all changes in physical conditions, such as better housing and sanitation, we cannot tell how long the improvements now being made will last, nor do we know whether they will tend automatically to promote any corresponding advance in the inborn qualities of the race. Even if we do not join in the vehement denial of the inheritance of acquired characters, now the attitude commonly adopted in the world of science, yet no one can in truth pretend to estimate the eugenic value, or the value to the generations of the more distant future, of any purely environmental reform. In order that we may stand on firmer ground we eugenists must continue to urge that further inquiry be made as to the biological effects of changes in human surroundings, an inquiry of the greatest difficulty. And, pending the result of such researches, we must continue to point out that if our object be to ensure the continued progress of the race, and if we wish to make certain that we are taking some steps in this direction, we must not confine our attention to reforms affecting environment. But where, then, are we to look if this be our quest? Is it not obviously to the factor of heredity, by means of which the inborn qualities of one generation are passed on to the next, to which we ought mainly to look when taking thought concerning the racial advancement of mankind? In fact, we have by a somewhat different route again reached the conclusion that heredity is the key by means of which the door leading to the future advancement of mankind must be unlocked.

But even if this view be adopted we have many difficulties to face before we can decide on the practical steps which should now be taken. Let us begin by imagining mankind first arranged in order according to the qualities held to be desirable, and then divided in two equal groups, when there would be one group composed of individuals all above, and another of those all below the average in what Galton called civic worth. This imaginary division having been made, it is no doubt evident that the main aim of the eugenist should be to decrease rate of multiplication in the lower of these two halves of humanity relatively to the rate of multiplication in the upper half; for the greater the success of such efforts, the greater would be the proportion of our descendants who would have sprung from superior stocks, and the more certainly would progress be thus ensured through the agency of heredity. But before the best methods of securing these results can be ascertained, many problems have to be solved, and much hard work must be undertaken. The first question which naturally arouses our attention is whether as a fact our nation is now advancing as regards its inborn qualities, or whether we are degenerating; that is, whether the less fit are multiplying so fast that they are, as it were, swamping the more fit, or whether, the reverse being the case, we are still on the upward path of racial progress. In order to decide in which half of humanity the rate of multiplication is highest, we must obviously have some method of sorting human beings into groups according to their innate qualities, or their biological fitness; and this we have now no means of doing. We can, it is true, generally say whether one group is richer or poorer than another; but does this afford any indication of their relative fitness? Or, in other words, is there any correlation between wealth and innate ability? Here is a problem urgently demanding our attention, to which but little thought has yet been given, and concerning which there is still much doubt. As a single example of the

way in which an answer to this question may be sought, what is being done by my society in this direction is perhaps worth mentioning. A scheme has been elaborated by means of which a record will, it is hoped, be obtained of the social classes, or the kind of surroundings from which certain boy and girl emigrants are drawn; whilst subsequently efforts will be made to follow their careers in the colonies. These young people go out without parents, and all under much the same conditions, the element of environment being thus more or less completely eliminated; and their innate qualities can therefore be judged by their subsequent success or failure in life. In this way we ought, therefore, in time to be able to ascertain whether these innate qualities do vary according to the social status of the emigrant's family.

If anyone can suggest any other and more ready method of ascertaining the inborn qualities of the different sections of the nation, we shall indeed be grateful; for, as it is, we have to rely mainly on *a priori* reasoning in considering whether there is any correlation between wealth and innate qualities. Certainly no one can be so foolish as to deny that there are amongst the poor many individuals both very virtuous and very capable, or that amongst the rich many most undesirable specimens are found, by whatever test they may be judged. But it is equally foolish to blind our eyes to the fact that a sifting process is in fact continually in operation; for the naturally strong and able amongst the poor often acquire greater wealth, whilst many of naturally deficient stock amongst the richer classes keep slowly sinking from their more comfortable estate. In fact, it can hardly be doubted that the poorest strata of society do contain an exceptionally large proportion of the biologically unfit; a fact which must fill us with grave alarm when we remember that statistical inquiries prove conclusively that this same section of the community has also an exceptionally high birth-rate. For if the unfit are

multiplying more rapidly than the fit, this we are certain must inevitably lead to national degeneracy.

But if the relatively low rate of multiplication of the better-off classes—I am not thinking about the very rich—is a source of danger to the nation, ought we not to consider carefully all possible causes which may account for this alarming fact? If we put aside the possible existence of any direct biological cause for the low fertility of the more cultivated classes, we yet have to inquire whether there are not certain factors acting in this direction in a more indirect manner. Almost everyone is considerably influenced by fears concerning the possibility of a diminution in his own comfort in the future, and also concerning the prosperity of any children he has or may have; and such fears inevitably tend to a postponement of marriage and a diminution in the size of the family. Moreover, such causes obviously affect the well-to-do more than the destitute; because the fear of a fall varies with the depth of the possible drop, those already at the bottom being troubled with no such apprehensions. The man in comfortable circumstances dreads the possibility of his children falling in the social scale, whilst no such thought can enter the head of the pauper. If this be the main cause of the comparative infertility of the more prosperous and presumably, on the average, abler classes, we have next to consider how this national disease may be cured.

In this quest our aim must obviously be, amongst other things, to make respectable parents with families look to their future with greater assurance than they at present feel. Possibly something might be done to increase the now somewhat slender confidence which is felt in regard to the contributions receivable from children in old age; with the result that the production of a family would seem to be less economically undesirable. Then again, it would unquestionably be possible to make the income tax vary substantially according to the

size of the family, and to deal with death duties in a similar way. Another proposal which ought, I think, to be adopted in this country, is that which has actually been tried at Schöneberg, near Berlin, namely, the adoption of a scale of pay for all government employees, dependent in certain measure on the number of their children; for, where none but good workmen are employed, this system would unquestionably be eugenic in its tendencies. In fact, there are several reforms to which the politician can devote his energies with confidence as to their beneficial eugenic results.

The expedients just mentioned for lightening the financial burdens of parenthood would, no doubt, touch but a small percentage of the nation, and more drastic remedies applicable to nearly the whole population are certain to be suggested. For example, a bill has been passed by the House of Representatives of Australia with the object of conferring a bonus of £5 to the mother of every child of white race at its birth. All such proposals must, however, be scrutinised with great care before they can be endorsed as regards their eugenic effects; for any system designed to increase the birth-rate generally may have the effect of increasing it more in the lower than in the upper half of humanity. If any proposed stimulus would be most felt by the least provident classes, as is probably the case with the Australian example just quoted, the results would almost certainly be dysgenic and not eugenic. In short, it seems that in our campaign we must not rely too much on such economic stimulants, for our main hope must rest on the possibility of changing the whole mental attitude of the nation in regard to the duties which it owes to posterity.

If the explanation of the low fertility of the well-to-do classes is that they pay too much regard to their future economic position, it may with equal truth be said that the high fertility of the destitute results from many of them taking but little thought of their future welfare; for innate mental

incapacity for forethought often leads to early marriages, large families, and poverty. Although something may be done to mitigate these joint evils by moral persuasion, yet it must be admitted that the prospects of beneficial results thus being obtained in the very lowest strata of society are but very slight. The man who is not in the least deterred from an improvident marriage by the prospect of immediate want is little likely to be open to eugenic or any other argument. A large class, in fact, certainly exists which cannot be touched by any such means, and it is within this class that the vast output of undesirable citizens is taking place which the eugenist is striving to lessen. How can this be done? Possibly some steps might and should be taken in the direction of making those who indulge in reckless marriages feel more immediately the unpleasant economic consequences thus arising; and this would be perfectly legitimate considering that by such marriages they inevitably throw a heavy burden on the shoulders of their more prudent neighbours in the form of increased poor-rates, etc. But if we look the facts of life fearlessly in the face we are driven to the conclusion that, for a large section of the community, the only methods by which the desired lessening of fertility can be obtained are sterilisation and segregation.

As regards sterilisation, I do not propose to discuss this subject to-night at length. In fact, I merely wish to express the opinion that whilst, on the one hand, this question should be approached with the utmost caution and in a most conservative spirit, yet, on the other hand, it would be well to watch carefully what is taking place in America. In eight states of the Union laws have been passed authorising sterilisation in the case of certain classes of degenerates; and, if little has as yet been done under cover of these laws, this may be due to a doubt as to whether they are constitutional, and therefore valid.

As regards segregation, this method of dealing with undoubted degenerates must now be regarded as well within the region of practical politics; for the eugenic arguments in favour of the Mental Deficiency Bill, though they aroused strong opposition, yet unquestionably carried much weight in many quarters. If we accept the explanation of the high fertility of the unfit already given—namely, that it is due to a certain deficiency on their part in mental powers—we should expect to find that families amongst the definitely feeble in mind are exceptionally large; and this is undoubtedly the case. The Eugenics Education Society had, therefore, had no hesitation whatever in placing the Mental Deficiency Bill in the forefront of their programme, and in promoting it by every available means. This they did by endeavouring to influence members of Parliament in the constituencies, by appearing before private committees in the House of Commons, by speaking at meetings, and by circularising the local authorities throughout the country. The victory has now been won, the bill having passed; but more work of this kind may at any moment become urgently necessary; work in which local eugenic societies could play a most useful part.

Besides semi-political agitation of this kind, serious scientific work is also needed in reference to the mentally defective. We want many more pedigrees of families containing feeble-minded individuals; and these must be collected with all the many precautions needed in such cases. These records are required in order not only to ascertain with greater accuracy the degree of probability of feeble-minded children appearing under given family conditions, but also to solve certain Mendelian problems. For example, is feeble-mindedness a simple recessive character? The asserted fact that when two feeble-minded persons marry, *all* their children are feeble-minded, points to this conclusion; whilst other facts are said to point to a different view. In

short, much still remains to be ascertained with regard to many genetic questions.

Eugenists ought, in fact, not to remain satisfied for long either as regards scientific inquiry or practical legislation with dealing with the feeble-minded alone; for there are many other classes of degenerates, notably criminals and paupers, whose multiplication ought to be diminished. In both these classes the trouble is, we believe, closely correlated with inborn mental defects, and more pedigree work is greatly needed in order to enable us to speak with greater confidence concerning the degree of probability of these characteristics being inherited.

Moreover, the whole question of psychological tests demands further attention. It is true that the Binet System has been carefully elaborated in France, by means of which it is held that criminals can be graded according to their mental capacity; whilst Prof. Münsterberg in his *Psychology and Crime* urges the value of measuring by definite experiment various physical attributes, such as accuracy of perception of touch, in connection with mental degeneracy. In fact, what we want to be able to do, as it seems to me, is to be able to mark out as a class apart those individuals who have been proved to be undesirable or defective as judged by *all* the three following tests:—

(1) In the case of criminals, by having been convicted of several crimes, the number depending on their gravity; and, in the case of paupers, by having been frequently in and out of the workhouse;

(2) By having been proved, either by evidence or by definite physical or psychological tests, to be either physically or mentally defective; and

(3) By having been reported by the prison or workhouse authorities, as the case may be, to be unable or clearly unwilling to gain an honest livelihood.

As regards criminals thus judged to be degenerates, they should be dealt with, not as needing punishment, but either as merely requiring either protection against themselves, or control for the purpose of safeguarding the public from present injury or future hereditary taint; that is to say, that they should be treated with great consideration, provided that their periods of confinement are never brief. As regards paupers coming within these three categories, much the same remarks are applicable; and, for their detention, labour colonies ought to be established in the United Kingdom in pursuance of the example set to us by Belgium and Switzerland. Neither with criminals, nor with paupers, should such detention in the first instance be regarded as being necessarily for life; for those whose failure can fairly be attributed in large measure to misfortune ought to have a chance of regaining their freedom in time. Even if a mistake is sometimes made in letting out a veritable degenerate, the evil will probably soon be remedied by his return to prison or to the workhouse. Such systems, in fact, must be judged by their results taken as a whole; and, as regards this proposal, it certainly would have the effect of materially reducing the output of defectives, which is all we can hope to do at present.

Eugenics is therefore, as we have seen, founded on the results of scientific investigation; and, in strengthening its foundations, much remains to be done. But since foundations standing alone are useless, what we have to do is to erect on them a moral superstructure; and here, I am convinced, our greatest difficulties will be felt. Our main object must be to sow broadcast throughout the nation a deep and impelling sense of our obligations towards posterity; a task which may be facilitated in many different ways. Eugenic education societies may be founded in various localities, a step which would prove, I believe, to be most beneficial; for everyone who by joining a society enrols himself as one of our army is quite

unconsciously thus induced to become an advocate in our favour in general conversation. Moreover, in definite propaganda work such local help is almost indispensable. For example, at Liverpool certain members of the council of the local eugenic society have done excellent service in themselves, giving elementary lectures in eugenics at various institutions, such as workmen's clubs, teachers' associations, etc. Again, such help is needed in making the best use of the local press. Lastly, especial efforts should be made to interest teachers in schools of all kinds in our cause; for, if the eugenic ideal could be properly planted in the minds of all children, not only would the effect be immediately ennobling, but good fruit would in time grow of itself out of this seed in the attitude adopted towards eugenic problems by the men and women of the future.

In conclusion, what I wish to urge most strongly is that it is not only the help of the scientist and the moralist that we need; for we appeal to every class of the community to join us in our fight against national degeneracy. If only it were more widely known—and here the economist could help us—how enormous is the burden now thrown on the community by the presence in our ranks of vast numbers of degenerates, then we should be better able to show how urgent is the need for reform. Increased taxation, often hidden in the form of increased rent; lowered wages; increased prices; diminished aid in times of trouble; and lessened power of competition in times of peace and of defence in times of war—these are the heavy penalties we are now paying for the ignorance and consequent carelessness of our ancestors with regard to the purity of our race. Are we to be equally careless as regards the future, and shall we pass on this taint with undiminished intensity to posterity? If ever we can succeed both in making the facts on which our creed is built more widely known, and in establishing as an accepted truth our belief as

to the grave danger of rapid national degeneracy, then I am certain we shall only have to appeal to our fellow-countrymen in the name of patriotism, in order to attract sufficient recruits to our ranks to make the success of our cause a certainty.

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